OFFICIAL PROGRAM

TRANSACTIONS

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of
OPHTHALMOLOGY
and
OTOLARYNGOLOGY

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1950

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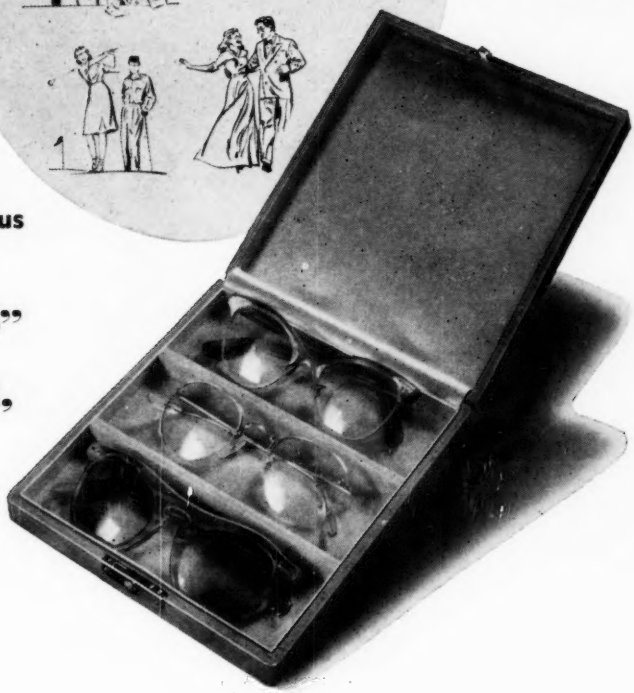

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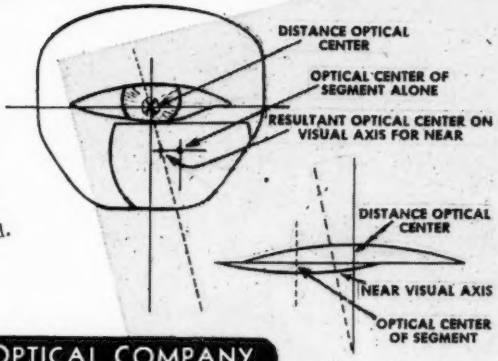
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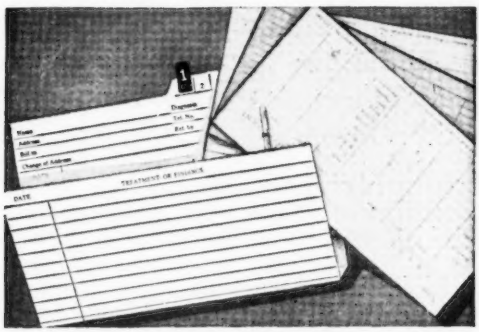

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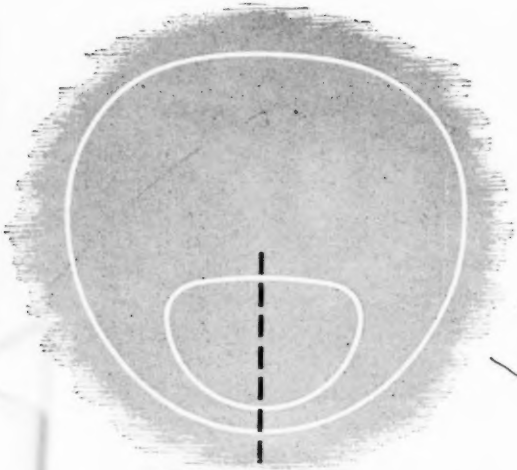

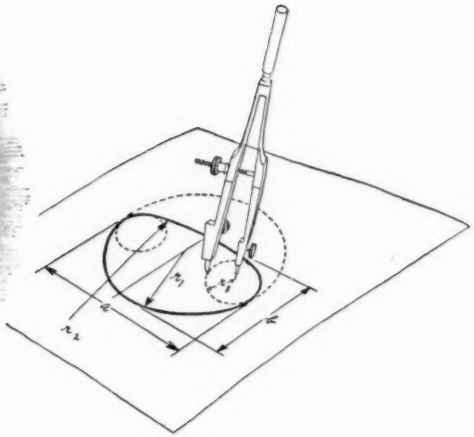

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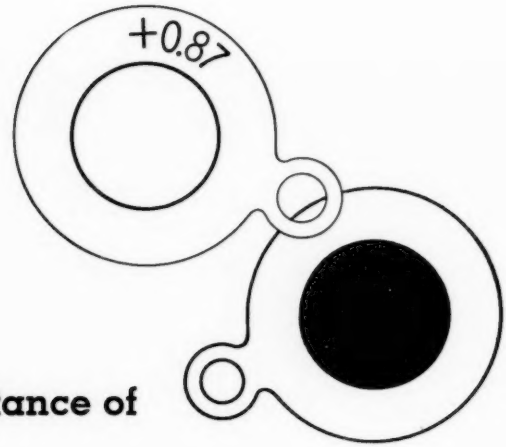

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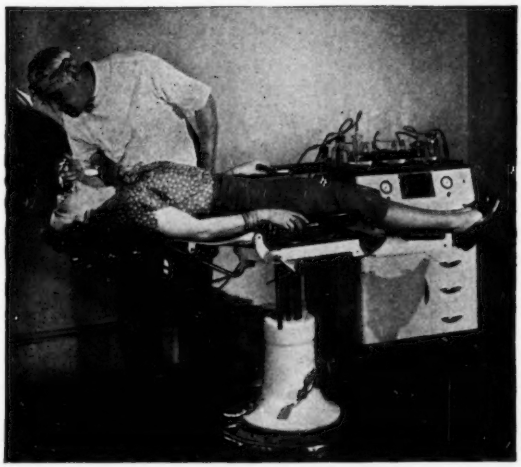

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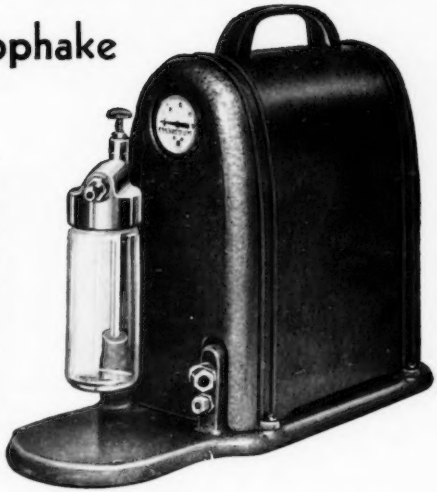

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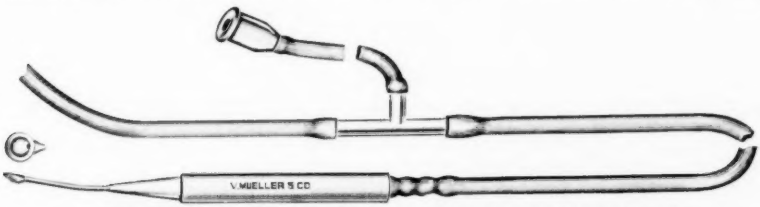

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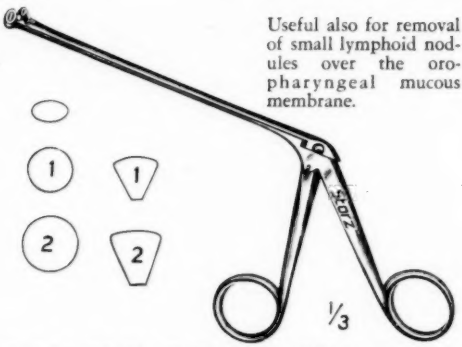

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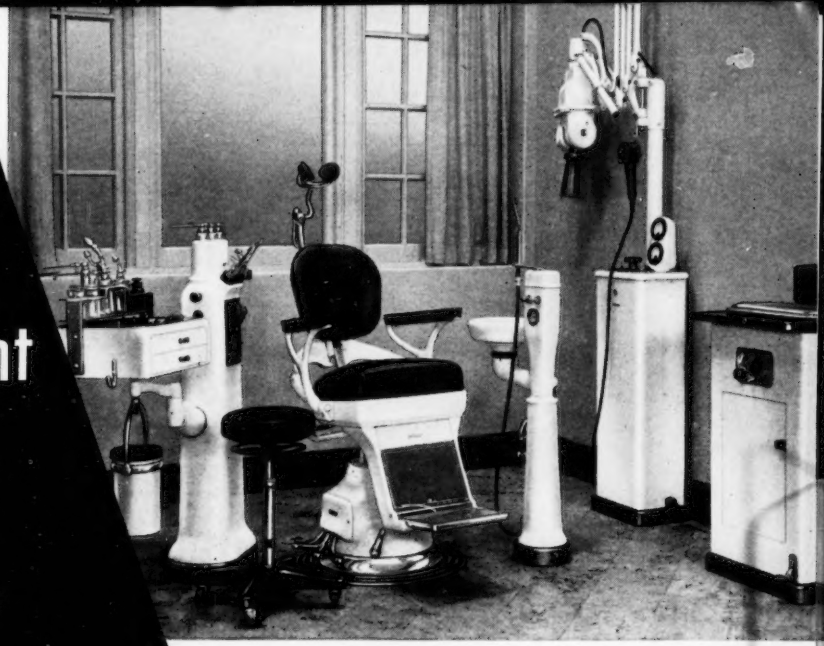

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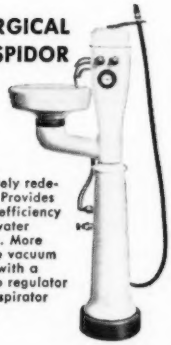

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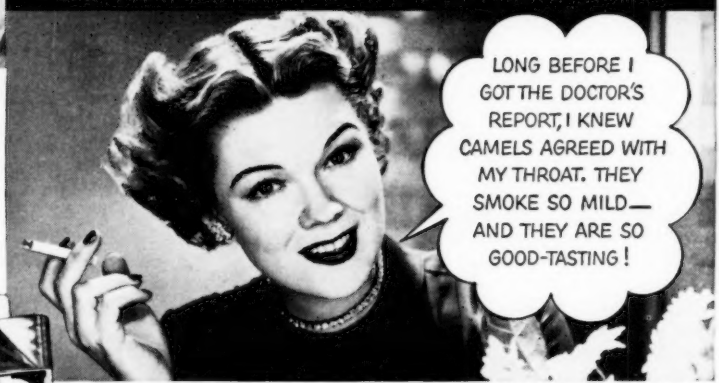

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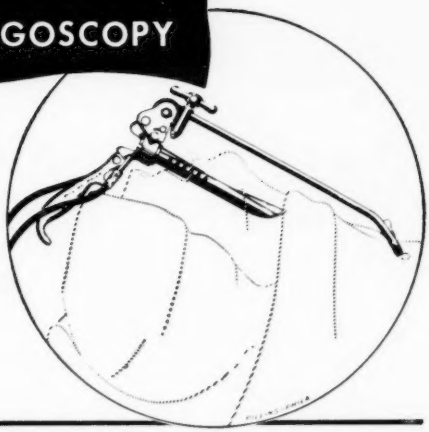

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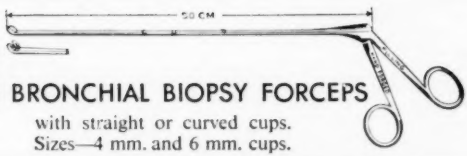

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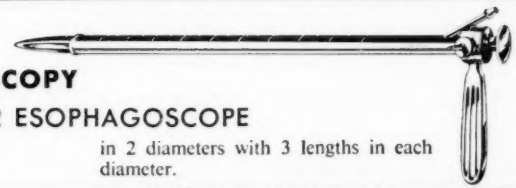

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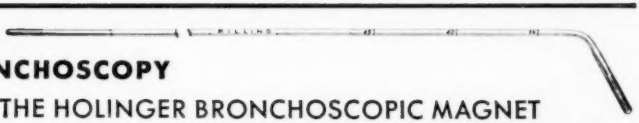

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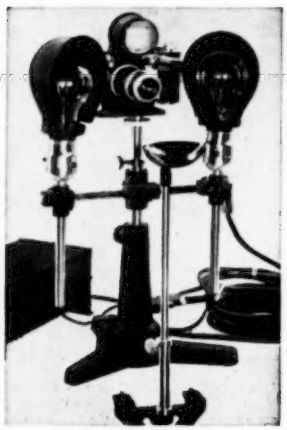

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CLASSIFICATION

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THE classification of primary chorioretinal aberrations with night blindness is, of necessity, an arbitrary compilation that most adequately conforms to the known relationships of the diseases to each other. Unfortunately precise pathologic knowledge is not available for all the conditions covered in the classification so that clinical characteristics must be utilized in formulating various divisions.

Congenital stationary night blindness is the essential attribute of the first group of diseases considered. The four entities falling into this class are physiologic aberrations that may be accompanied by slight structural change, but they are not diseases that ultimately cause destruction of vision. For the most part they are recessive in hereditary character, and it is probable that they constitute variations of the same entity. Such variations are well illustrated by Nakamura's description of three forms of Oguchi's disease in which variations in the appearance of the fundus and difference in the degree of secondary dark adaptation differentiate the types. The three types of Oguchi's disease are not included in the outline because doing so would seem to call too much attention to a disease of its rarity.

From the department of ophthalmology, College of Medicine, State University of Iowa.
Presented at the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1949, Chicago, Ill.

The recognition that retinitis punctata albescens may exist both as a stationary and a progressive type appears to be justified.

The second division consists of those diseases in which night blindness is delayed in onset and progressive in character. Retinitis pigmentosa is the most important disease in this group. It occurs alone or associated with a number of ocular and systemic defects ranging from myopia to the mental deficiency, obesity, polydactyly, and genital dystrophy of the Laurence-Moon-Biedl syndrome. Myopia occurs in approximately 50 per cent of the patients with all types of retinitis pigmentosa, but it is a constant manifestation in the sex-linked recessive form. Systemic defects occur predominantly in those patients with recessive heredity. Retinitis pigmentosa sine pigmento is not included as an entity because of the great variation in the amount of pigment that occurs in different cases and because evidence indicates that the unpigmented state represents an early stage of the fully pigmented form.

The final group is composed of those diseases in which atrophy of the choroid is the essential characteristic. The term choroideremia is retained, although its deficiencies are realized, because a new term introduced at this time might be no more exact and would serve no useful purpose.

PRIMARY CHORIORETINAL ABERRATIONS WITH NIGHT BLINDNESS

I. Congenital Stationary Night Blindness
- A. Without ophthalmoscopic abnormality
  - Dominant autosomal heredity
  - Recessive autosomal heredity
- B. Without ophthalmoscopic, but with other, abnormalities (myopia, amblyopia, nystagmus)
  - Recessive sex-linked heredity
- C. Ophthalmoscopic appearances characterized by changes in color of the fundus. *Oguchi's disease*
  - Recessive autosomal heredity
- D. Ophthalmoscopic appearances characterized by white dots in the fundus. *Retinitis punctata albescens (stationary type)*
  - Recessive autosomal heredity

II. Delayed and Progressive Night Blindness
- A. Ophthalmoscopic appearances characterized by white dots in the fundus. *Retinitis punctata albescens (progressive type)*
  - Recessive autosomal heredity
- B. Ophthalmoscopic appearances characterized chiefly by pigmentation of the retina. *Retinitis pigmentosa*
  - 1. Without significant associated defects
    - Dominant autosomal heredity
    - Recessive autosomal heredity
  - 2. With associated defects
    - a. Ocular
      - Myopia
        - Sex-linked recessive heredity
      - Atypical chorioretinal changes
    - b. Systemic
      - Deafness and deafmutism
        - Recessive autosomal heredity
      - Mental deficiency
        - Recessive autosomal heredity
      - Other neurologic disturbances (ataxia, paraplegia, ophthalmoplegia, etc.)
      - Laurence-Moon-Biedl syndrome (mental deficiency, obesity, polydactyly, genital dystrophy)
        - Recessive autosomal heredity
- C. Ophthalmoscopic appearances characterized by depigmentation of the fundus. *Progressive atrophy of the choroid*
  - 1. Gyrate atrophy type
    - Recessive autosomal heredity
  - 2. "Choroideremia" type
    - Intermediate sex-linked heredity
    - a. Male form
    - b. Female carrier form

SYMPTOMS, SIGNS AND DIFFERENTIAL DIAGNOSIS

FRANK D. CARROLL, M.D.
NEW YORK, N. Y.

(Note: This discussion will follow the order proposed in our classification.)

CONGENITAL stationary night blindness without ophthalmoscopic or other abnormality has the following characteristics: (1) the condition is present at birth; (2) it is stationary; (3) it occurs in an approximately equal number of males and females; (4) no other abnormalities are present. In daylight the visual acuity and fields are normal. At night, or in reduced illumination, visual acuity is markedly reduced and visual fields greatly contracted. This disease, though apparently rare in this country, was not uncommon in certain localities in France[21] and elsewhere. Nettleship states[22] that mothers, fearing their children might be night-blind, tested them from early infancy by holding objects for them to see in reduced illumination. These mothers could detect night blindness while infants were still very young. Children with this disease frequently walk at night or in dark places with their arms extended as though they are blindfolded. When older, they distinguish in the dark large objects, such as buildings, by the sound of their own footsteps or air currents being reflected from these objects. On clear nights they are guided along streets by watching the outlines of the buildings or trees against the sky. But nearly all persons with night blindness do have minor or major accidents in the dark. Dark adaptation studies[6,16] indicate that the retinas of such patients have no functioning rods. The cone curve appears normal, but the rod curve is entirely absent. The amount of dark adaptation possible by cones alone is so slight that the older writers stated that these patients showed no dark adaptation at all.[22]

However, this is a relatively mild condition compared to congenital stationary night blindness with other abnormalities,[24] which occurs only in males. Myopia occurs in this group, but even with correction the vision is subnormal. Nystagmus is occasionally present. The fundi appear normal except for changes associated with the myopia, but these changes do not account for the amblyopia. The myopia frequently remains stationary for years. Mental defects are common in this group.

It is important to consider such rare diseases as this because they give us a background for understanding more common diseases like retinitis pigmentosa.

The next link in this group is a type of so-called Oguchi's disease. Oguchi[25] described cases of stationary night blindness which showed a discoloration of the fundus. Mizuo demonstrated that in many of these patients the discoloration disappeared after the patients were in the dark a few hours: this is called Mizuo's phenomenon. Nakamura[20] in

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doing dark adaptation tests found that one and a half to two hours after cone adaptation, rod adaptation did occur: in other words, there was secondary dark adaptation. However, some cases show only slight discoloration with no disappearance of the discoloration in the dark and no secondary dark adaptation; some show disappearance of the discoloration when the patient is placed in the dark but no secondary dark adaptation. These are probably transition types between congenital stationary night blindness with no fundus changes and Oguchi's disease with marked discoloration of the fundus, Mizuo's phenomenon and secondary adaptation. Such is the case reported by Dr. Klien[12] of Chicago. She states, "A wavy yellowish white discoloration surrounds the disc and macula and extends well into the periphery where a gradual transition into normal fundus could be observed." The retinal vessels seem darker than normal against the light background. Dark adaptation is markedly delayed. Dr. Klien placed the patient in the dark and examined the fundi every hour; at the end of four hours all the discoloration had gone except for one small spot which had been most dense originally. The patient had been night-blind all her life. Usually the visual acuity and color sense are normal; the fields are normal in daylight but contracted in reduced illumination.

Retinitis punctata albescens is a term which Nettleship[22] felt "should be restricted to the cases in which the fundus is sown pretty evenly with innumerable discrete white dots entirely free from pigment . . . apparently dating from birth and remaining stationary." Night blindness is present from earliest recollection. Fields are contracted only in reduced illumination. Dark adaptation study of one patient has shown a normal cone function but no rod function.[14] The dark adaptation curve of another patient showed "a marked delay in adaptation, but after thirty minutes the subject reached a threshold level reached by a normal person in twelve minutes."[17] This delay in dark adaptation would help explain the clinical observation of Nettleship.[22] His patient, after being in the sun, was unable to see a man sitting beside him for thirty to sixty minutes. The patient claimed he had no trouble with night vision. Nettleship followed 1 patient 27 years and found no change in her fundi. Another patient was followed 18 years without change, but eight years later there were remarkable changes. All the white dots had disappeared, bone-corpuscle shaped pigment patches had formed and marked choroidal sclerosis was present. The characteristic appearance of retinitis punctata albescens changed to that of retinitis pigmentosa. Therefore, we must conclude that these cases may be *stationary* or *very slowly progressive*. Lauber[13] describes a progressive type with pigmentary changes, narrowing of the retinal vessels, and constricted visual fields: this resembles retinitis pigmentosa plus the white dots, and we classify this condition under delayed and progressive night blindness.

The signs in retinitis pigmentosa are well known, that is, pigmentary degeneration of the retina, narrowing of the retinal vessels, waxy appearance of the disc, loss of visual field, and frequent development of cortical lens opacities. The pigment distribution is frequently along the vessels and in the shape of fine branching figures described as bone-corpuscle shaped or spider-like formations, but besides these figures there are usually irregular accumulations of pigment. This distribution of pigment is first located in the midperipheral region, but as the disease progresses it spreads both centrally and peripherally, corresponding to the loss of visual field. The choroidal vessel markings may be in-

conspicuous or may be very prominent, and the macula may exhibit a shiny appearance. One of Dr. Perera's patients[26] has a hole in the macula, a rare complication. This patient has been followed 12 years since the hole developed in the macula of one eye; the visual acuity of the other eye is normal and the macula appears normal. When central vision is impaired early in the disease, the patient, instead of being night-blind, sometimes prefers a dull light. Dark adaptation studies[10] also indicate that in some mild cases the central area may show greater functional impairment than certain peripheral areas.

The decrease in caliber of the retinal arterioles is a very gradual process which may continue until these vessels are barely visible. The disc is often normal in appearance or may appear "waxy" in color. Posterior and occasionally anterior cortical lens opacities frequently develop. Often the disc must be viewed through these opacities and the apparent color and outline of the disc depends in part on the media through which it is viewed. The term "waxy" as used in connection with the appearance of the disc seems much overworked. Frequently the disc appears normal until late in the disease. Walsh,[33] however, mentions 1 patient with so much gliosis of the disc that the appearance resembled papilledema.

Dark adaptation studies indicate a gradual decrease in the function of the rods until only the cones are being used. The first visual field defects usually occur in the midperiphery as irregular scotomas whose boundaries are difficult to outline. Test objects come and go in this area. Then these scotomas merge into a partial or complete ring scotoma. This frequently extends from the 10 degree circle to the 40 degree circle. In dim light the field loss is much greater, especially the peripheral depression. The ring scotoma gradually enlarges, breaks through to the periphery and comes closer to the point of fixation. The last part of the peripheral field to remain is frequently a crescent shaped area temporally. Traquair[31] mentions the steep edges of the small central field. Even a very small posterior cortical lens opacity may cause considerable impairment of the visual acuity in a patient who has a markedly contracted visual field. Glaucoma is a complication which occurs in 1 to 3 per cent of the cases.

Sixteen cases of unilateral retinitis pigmentosa have been reported. One of them was followed ten years, another six years[1]; the condition progressed in the involved eye while the other eye remained normal. Although usually the disease is apparently the same in the two eyes, one eye may show signs of the disease years before the other eye is involved. Sometimes there is loss of field in one eye long before the pigmentary changes become apparent. Occasionally one eye shows typical retinitis pigmentosa and the other only reduced dark adaptation with a normal field and fundus. Retinitis pigmentosa sine pigmento usually indicates merely an early stage of the disease. The loss of field may be entirely out of proportion to the amount of visible pigment disturbance. Numerous cases,[23] however, are recorded, some of them in families where other members showed the typical pigmentation. The fields may be contracted almost to the point of fixation, the visual acuity may be impaired, but no pigment disturbance is seen.[2]

Myopia occurs in approximately 50 per cent of patients with retinitis pigmentosa, but Usher[32] reported a pedigree in which all affected members had myopia and all were males.

Patients showing numerous atypical chorioretinal changes are not infrequent, and many cases have been collected by Gordon.[9]

Deafness or deaf mutism is very frequently an associated defect in certain families. The two conditions, deafness and retinitis pigmentosa, occur almost inseparably in some pedigrees.

Mental deficiency was present in 4 per cent of Julia Bell's whole series,[2] but here, also, the defect is much more common in certain pedigrees.

Numerous neurologic conditions may be found. Walsh[33] mentions epilepsy, progressive muscular atrophy, spastic paraplegia and quadriplegia; chronic polyneuritis and cerebellar disease may be present.[27]

The Laurence-Moon-Biedl syndrome consists of the association of atypical retinitis pigmentosa with obesity, polydactyly, hypogenitalism, and mental retardation. There is great variation in the fundus picture in this condition. Typical retinitis pigmentosa may be present, or macular changes may dominate the picture. These may resemble those found in cerebromacular degeneration. Retinitis punctata albescens may occur, but most commonly there is seen some type of pigmentary degeneration sparing the macula. The symptoms usually begin in early childhood and progress very gradually.[29] Of the five features of the complete syndrome, that is, retinal degeneration, obesity, hypogenitalism, mental retardation, and polydactyly, only the first two are constantly present. The obesity may be lost as the child grows older.[5,30] A boy with this syndrome has been followed in our clinic for seven years. Besides the pigmentary macular degeneration and night blindness, he had obesity and hypogenitalism. Now at the age of 18 he has only the retinal degeneration. Large drusen are evident on the disc. I have also seen these in retinitis pigmentosa and choroideremia. In this case the drusen developed during the year 1944 and have increased in size since then.

Gyrate atrophy of the choroid and retina is a definite progressive disease. Night blindness is usually present from early childhood. A young man with this condition has been under observation for twenty years; he was 11 years old when the late Dr. Hunter McGuire[18] first examined him. At that time numerous fine granular changes were seen in the midzone, and in the extreme periphery was a circle of irregularly shaped atrophic areas. Nine years later the atrophy had progressed considerably.[19] At the age of 29 the patient underwent bilateral cataract extractions by Dr. Dunnington. The visual field is 5 degrees around the point of fixation in each eye, and there is a 5 degree ring of visual field 30 to 35 degrees from the center. Visual acuity with correction is 20/50, 20/60.

Recently Saebo[28] reported four brothers with this condition. All have been night-blind since childhood. The youngest, age 27, has normal visual acuity; the next two older brothers are considerably worse, and the oldest, age 46, has only light perception. The scalloped pigmented border of the atrophy in the latter case is characteristic. The fundus just peripheral to the expanding border is grayish. Finally, there remains only a thin rim of normal appearing choroid in the far periphery.

Most of these patients have myopia; the retinal vessels are usually reported as being contracted; a posterior cortical cataract is sometimes present; fundus pictures indicate the lobulated areas of atrophy which gradually spread both peripherally and centrally. Visual fields usually show a progressively enlarging ring scotoma.

CHOROIDEREMIA

The females, who are carriers and who have no apparent visual symptoms, have definite eye changes which are limited to the fundi.[15] Pigment mottling, usually more pronounced in the midperiphery and periphery, is the outstand-

ing sign. This pigment resembles coal dust; it is not in the shape of bone-corpuscles or spider webs; its location is posterior to the level of the retinal vessels, and it seems to have no special relation to these vessels. Occasionally in the periphery the pigment may group itself into a radial linear arrangement, but more often there is an irregular distribution of small clumps scattered throughout the fundus. This usually spares the macula, but occasionally this area is also involved although the vision is normal. Areas of depigmentation, probably of the pigment layer of the retina, are frequently present so that, especially in the periphery, choroidal vessels are visible. Small grayish white spots are often scattered throughout the areas showing the widespread pigment disturbance. The McCullochs have found no progress in the fundus changes in the females: they feel it is a congenital state which is stationary. It is no more marked in adults than in children: an adult may show only slight changes; a child, a much more striking picture. Several patients had peripapillary areas of depigmentation, but all had normal appearing discs and retinal vessels.

The earliest changes in the males closely resemble those seen in the female. Several boys had the typical female fundus plus areas of choroidal degeneration in the midperiphery. In their teens these young men showed larger grayish white areas and some had the typical adult male picture. When the oculist first begins his ophthalmoscopic examination of the male, he is struck by an amazing sight — a grayish white fundus reflex instead of a red reflex. If a special effort is made to direct the ophthalmoscope light at the macula, a red reflex is often obtained from this area except in the most advanced cases. The lenses in older patients may show posterior cortical or other lens opacities similar to those found in retinitis pigmentosa, but thus far the McCullochs have not had any case requiring cataract extraction. The vitreous may contain fine pigment particles or other opacities as occasionally seen in retinitis pigmentosa, but these have not appreciably obstructed vision.

The disc and retinal vessels, except in the most advanced cases, appear normal. Patients in their sixties and seventies may show narrowing of their retinal vessels, probably due to arteriosclerosis, and the discs may be pale and show loss of substance, but the striking thing about the disc and retinal vessels is how normal they appear despite the widespread pathologic change present. The choroidal degeneration seems to extend from the midperiphery both centrally and peripherally. Frequently a man has an area of red fundus from the disc to and including the macula and patches of red fundus in the far periphery. Then the red color near the disc is lost, leaving only the macula area intact. Even when the macula area decreases to a spot at the fovea, there may be some pink tissue seen in the far periphery. Choroidal vessels branch out from the macula area and occasionally extend to the peripheral areas, sometimes to a vortex vein. Choroidal vessels frequently run between the peripapillary area and the macula, and the vessels often show sclerosis. They bend and disappear into a white background. Infrequent clumps of pigment are seen against the light background. They are located at a level posterior to the retinal vessels.

Visual fields correspond fairly well to what would be expected—first, scotomas in the midperiphery or a ring scotoma in this region, then an extension of the defect centrally and peripherally. Next, all peripheral vision is lost; the central field frequently stops at 5 to 10 degrees for many years, but finally this, too, may be lost. If the fields are

examined in reduced illumination, a much larger defect is found in the early stages.

The visual acuity remains good ordinarily until the field contracts to about 15 degrees; then it also gradually decreases, but frequently the man retains 20/200 vision or better until in his fifties. Sometimes, however, vision may be this poor even in the teens. Transillumination through the sclera allows the light to come through all around the globe, except in the region of the ciliary body. Nerve deafness occurred in 10 of the McCullochs' 86 cases, but this was not necessarily considered as an associated hereditary defect. Myopia was very frequent in the McCulloch cases.

DIFFERENTIAL DIAGNOSIS

Congenital night blindness without ophthalmoscopic abnormality may be differentiated from vitamin A deficiency by (1) the family history in the former and the nutritional history in the latter; (2) dark adaptation tests[14] — in congenital night blindness there is no rod curve: in vitamin A deficiency both the rod and cone threshold are increased; (3) treatment with vitamin A usually helps night blindness which is due to its deficiency.

Congenital night blindness without ophthalmoscopic abnormality may be differentiated from retinitis pigmentosa sine pigmento by the presence of partial or complete ring scotoma in the latter condition.

Retinitis punctata albescens may be differentiated from vitamin A deficiency with fundus changes — a condition described by A. Fuchs.[8] He calls this transient retinitis punctata albescens because the innumerable white dots disappear soon after recovery from the night blindness.

Primary retinitis pigmentosa must be differentiated from numerous secondary degenerations. These include lues, trauma, measles and German measles, vaccinia, scarlet fever,[4] malaria, and typhus[3] in the patient, or German measles in the patient's mother during pregnancy.[11] Syphilis may produce a fundus picture which is very difficult to distinguish from retinitis pigmentosa. The visual fields and night blindness may be very similar. This occurs especially in the later stages of luetic neuroretinitis, in disseminated chorioretinitis, and in congenital syphilis. Frequently, however, in lues the pigment is not associated with the vessels and does not show the fine branching figures characteristic of retinitis pigmentosa. Also in lues, Fuchs states, the chorioretinal lesion may extend to the far periphery, the night blindness is not so marked, the disc is whiter, and ring scotomas are usually absent.

The loss of vision following the exanthematous diseases mentioned usually comes on rapidly and the fundus is left with an appearance resembling retinitis pigmentosa. There is a recent article from Australia reporting the fundus appearance of atypical retinitis pigmentosa in some children whose mothers had German measles during pregnancy.[11]

The fundus of retinitis pigmentosa patients with macula involvement, such as is often seen in the Laurence-Moon-Biedl syndrome, may simulate the fundus seen in the hereditary macular degenerations, including juvenile amaurotic family idiocy. But the progressive mental deterioration in the latter condition is characteristic.

Early cases of gyrate atrophy and choroideremia may resemble each other ophthalmoscopically, but in gyrate atrophy the scalloped edges of the atrophic areas are characteristic. Werkle[34] diagnosed his case as gyrate atrophy, but it lacks the scalloped edges and has the typical appearance of an early choroideremia. McCulloch has

shown me numerous young males with choroideremia who have fundi resembling this. Finally, the type of inheritance is entirely different in the two conditions.

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GENETICS

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RETINITIS pigmentosa and the allied diseases we are discussing today may be considered as hereditary even where proof is lacking or a superficial survey fails to reveal its incidence in the relatives of the patient.

An interesting and almost specific human genetic phenomenon is exhibited by these diseases in that the same clinical disease may present different inheritance modes in different families. From the eugenic standpoint it is therefore evident that no reliable advice can be given to any afflicted individual until the nature of his or her specific pedigree is carefully worked out. It is also prerequisite that a careful examination of the reported normal individuals as well as the afflicted members of the family be performed in order to evaluate carefully the clinical variations and modifications of the disease. Then and only then will the ophthalmologist or the geneticist have sufficient evidence upon which to base a prognostication of the mode and possibility of transmission of the affliction.

MODERN GENETICS

Since it will be necessary to use a terminology that is probably foreign to many in the audience, time will be spent in providing a common background.

The laws of heredity are fundamentally simple and concise, yet their comprehension necessitates diligent study and application. The recent and rapid advances in genetic theory and methodology have introduced complexities which make the understanding of genetic principles, on the part of the clinician, a difficult task.

The modern conception of hereditary transmission of traits is based on the "gene theory." These genes are minute structural units (complex protein molecules) arranged in single file order on the chromosome. A chromosome is comprised of hundreds of separable genes each having a fixed place in the above mentioned order. The gene possesses an individual and specifically distinctive internal composition. The individual gene is believed to play a very specialized role (or roles) in governing the production of the character or trait. It is assumed that the genetic (or gene) action takes the form of a chemical influence on the rate or direction of a process of development, of synthesis or of degeneration. Theoretically it is conceivable that these influences are effected through either enzymatic or antigenic action. It is most convenient to associate one gene with one character or trait, hence the conception of units. In reality this is not so, as most traits are the product of the interaction of many genes. A specific gene may be the principal factor in the production of an aberration.

No geneticist will present the view that the gene is always the cause of the trait. The great majority of ocular char-

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acters are actually fashioned by the combined or dual action of environment and heredity (genotype). This duality in causation makes for the complexity of clinical genetics. The gene is simply an impulse toward development in a specific way, but the influence of neighboring tissues, blood-borne toxins, serology, chemicals and abnormal nutrition may certainly modify the end product.

PATTERNS OF HEREDITARY TRANSMISSION

While there are many known patterns of hereditary transmission, we shall only consider the major kinds of genetic behavior, such as autosomal dominant, autosomal recessive, and sex-linked.

In the human species there are 48 chromosomes comprising 24 pairs; of these, one particular pair is different from the others and has a special relation to sex. Any chromosome other than the sex chromosome is called an autosome; all the remaining 46 may thus be referred to as the autosomes.

A. *The Characteristic Features of Autosomal Dominant Inheritance*

Requisites

1. Every affected person has an affected parent.
2. An affected person may be expected to transmit the abnormal trait to approximately 50 per cent of his or her offspring.
3. The trait is manifested (expressed) successively generation after generation.
4. Normal members of the family cannot transmit the abnormal gene since they do not possess it.

This pattern of inheritance is the most dramatic and elicits attention because of its obviousness.

B. *The Characteristic Features of Recessive Inheritance*

Requisites

1. An affected individual (homozygous) must have received the abnormal gene from both parents (heterozygous).
2. Neither parent manifests the trait, but being heterozygous they are both "carriers" of the abnormal gene.
3. The more rare the trait, the higher the proportion of consanguinity among the parents over that found in the general population.
4. When two affected individuals mate, all of their children will exhibit the trait.
5. Approximately 25 per cent of a sibship (brothers and sisters) may exhibit the defect (familial).

The important factor in recessive inheritance is that the affected individual must have received the gene for the trait from both parents; thus both members of the chromosome pair must carry the abnormal gene at the same locus. The rarer the abnormality, the more likely it is that the affected children will have resulted from consanguinous unions. Cousins (and like relationships) are individuals who will have received, in common, chromosome material (genes) from the same pair of grandparents. This chromosome material is therefore most likely to contain the same recessive genes. Recessiveness of a character is most difficult to prove.

C. *The Characteristic Features of Sex-Linked Recessive Inheritance*

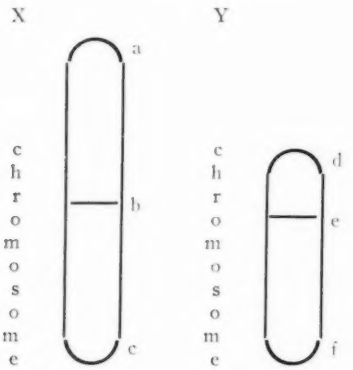
Requisites

1. Affected individuals have normal parents.
2. Heterozygous females (carriers) have affected and normal sons in approximately equal proportions.
3. Heterozygous females have carrier and normal daughters in approximately equal proportions.
4. The affected male cannot transmit the abnormal gene to his male offspring (unaffected Y is transmitted only).

5. All daughters of an affected male will be carriers (heterozygous).
6. The gene is transmitted through carrier females and is expressed in males.
7. Unaffected males cannot transmit the gene.
8. Females rarely display the trait since it is necessary to have received the gene from a heterozygous mother and an affected father.

In this type of inheritance we are dealing with a pair of chromosomes having a special relationship to sex. The abnormal gene is recessive and is borne on the X-chromosome. A pedigree of sex-linked inheritance is highly characteristic and nearly as dramatic as a pedigree of dominant autosomal inheritance.

Incomplete sex-linked dominant and incomplete sex-linked recessive inheritance patterns are also known and well warrant the following discussion.



1. *The X-borne genes.* Many genes are carried on the larger portion of the X-chromosome, a-b in the diagram. In the females there are two X-chromosomes. An individual female may be homozygous or heterozygous for a specific gene. In the case of the male there is only one X-chromosome and the Y-chromosome has no portion corresponding to the region a-b in the X-chromosome. The male can, therefore, never possess two genes at the same locus but only one. Thus there is no dominance or recessiveness of a gene in the male in the case of X-borne genes. The single gene will always be expressed in the male no matter what role it plays in the female.

2. *The Y-borne genes (d-e).* These genes are exclusive to the male. No recessiveness or dominance exists since when a gene is present here it is always expressed. No known examples of Y-borne ocular disease are yet known.

3. *X- and Y-borne genes (b-c, partial or incomplete; e-f, sex-linkage).* These regions on the X and Y chromosomes (b-c) and (e-f) are homologous and genes carried in these areas may be present on either the X or Y or both chromosomes. They always occur in pairs just like the autosomal genes, and like the latter may be recessive, dominant or intermediate.

INHERITANCE PATTERNS EXHIBITED BY THE PRIMARY CHORIORETINAL ABERRATIONS WITH NIGHT BLINDNESS

I. *Congenital Stationary Night Blindness*

A and B. This disease state presents three specific patterns of inheritance: dominant autosomal (fig. 1), recessive autosomal (fig. 2), and sex-linked recessive (fig. 3). The latter pattern of inheritance presents an association with myopia, nystagmus and occasionally amblyopia. Other than for the different genetic pattern there is no specific clinical distinction between the dominant autosomal and the recessive autosomal forms of the disease.

C. *Oguchi's disease.* This disease is inherited as a simple autosomal reces-

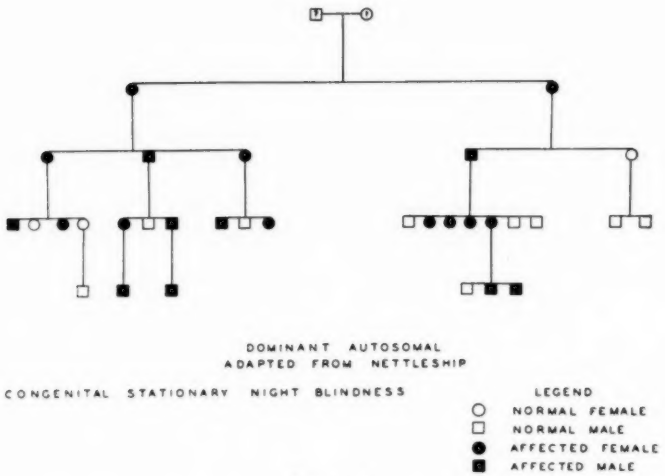


FIGURE 1.

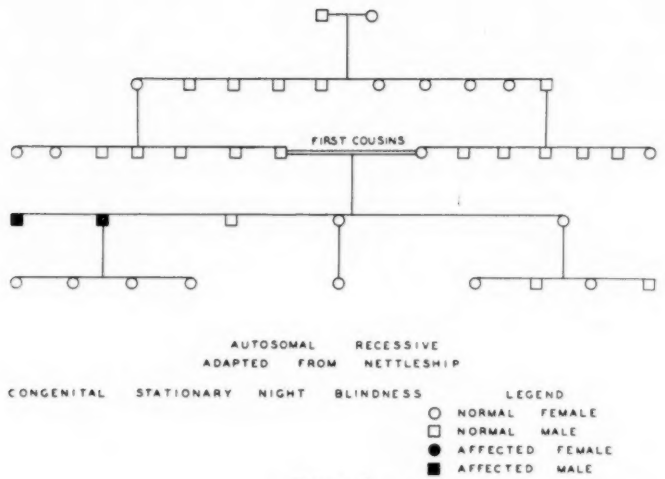


FIGURE 2.

sive (fig. 4). It occurs in brothers and sisters. The parents of these afflicted individuals present a very high incidence of consanguinity. J. B. S. Haldane[5] has postulated an incomplete sex-linked recessive inheritance for Oguchi's disease.

D. *Retinitis punctata albescens.* There apparently are many forms of retinal aberration which are considered as punctata albescens. When one, however, restricts consideration to those cases of retinal degeneration characterized by

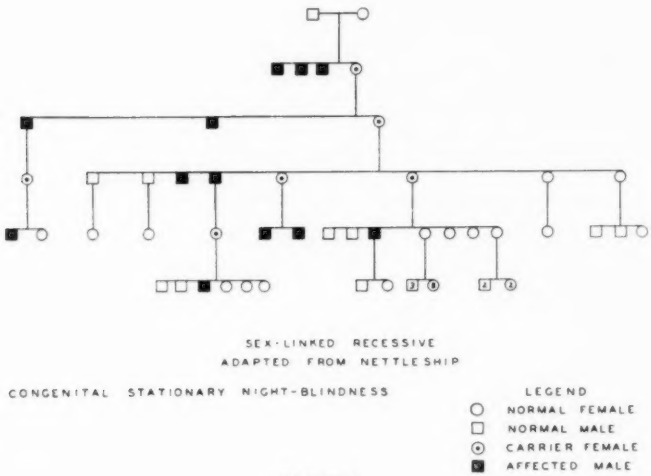


FIGURE 3.

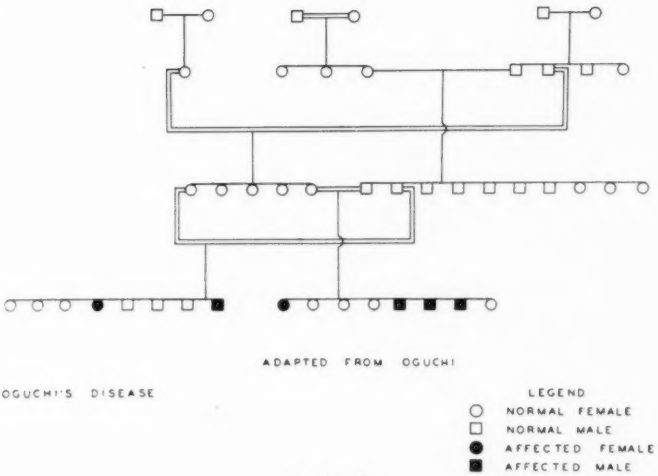


FIGURE 4.

the presence of innumerable white discrete dots scattered over the entire fundus with or without only minimal pigmentary changes, then a specific hereditary pattern becomes evident (fig. 5). The disease is apparently inherited as an autosomal recessive trait. The disease occurs among brothers and sisters. There is a high incidence of consanguinity among the parents of such families. Retinitis pigmentosa has been observed in the same sibships. A. Sors-

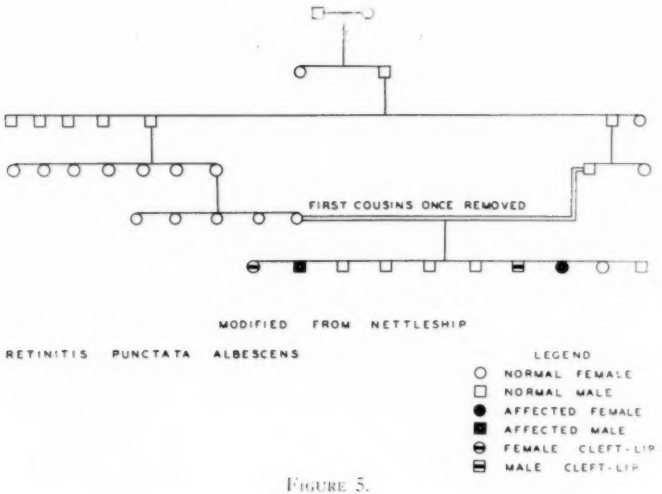


FIGURE 5.

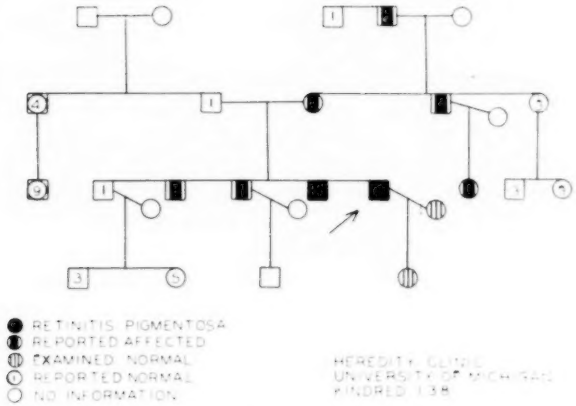


FIGURE 6.

by[7] feels that "the whole group of Retinitis Punctata Albescens is hodgepodge that requires detailed study."

II. *Delayed and Progressive Night Blindness*

A. The progressive type of retinitis pigmentosa has been discussed under I-D.

B. *Retinitis pigmentosa.* Retinitis pigmentosa presents a great variation in degree, onset, course and prognosis even within the same family. A greater range of variation and expression occurs between unrelated families. Once cognizant of these facts we might anticipate that there are a number of different

genes which can produce nearly similar clinical pictures. Actually such is the case, for today students of genetics recognize five different modes of hereditary transmission of indistinguishable clinical retinitis pigmentosa: (1) dominant autosomal, (2) recessive autosomal, (3) sex-linked recessive, (4) incomplete sex-linked dominant, and (5) incomplete sex-linked recessive. A possible sixth mode of intermediate sex-linked inheritance will be mentioned.

1. Dominant autosomal (fig. 6). The literature is replete with incomplete pedigrees showing only second or third generation transmission of retinitis pigmentosa. To be certain of a dominant mode of inheritance we must have three or more generations of afflicted individuals. It is extremely rare to encounter mental deficiency or deafness in dominant pedigrees. Actually when such (mental deficiency or deafness in dominant pedigrees) is encountered, explanations other than an association with retinitis pigmentosa should be considered. As stated by A. Franceschetti:[3]

> When an association between dominant retinitis pigmentosa and deafness or mental deficiency is encountered, this should be explained as due to an accidental manifestation of two genes rather than a polyphenous expression of one gene, as would be the case for recessive retinitis pigmentosa combined with the same anomalies.

2. Recessive autosomal (figs. 7 and 8). This is the most common form of hereditary transmission. There may actually be many genes producing autosomal recessive patterns of retinitis pigmentosa. At least two are known.

a. A high proportion of the pedigrees show an association with deafness or deafmutism.

b. Many pedigrees show a high incidence of mental deficiency or other central nervous system aberration.

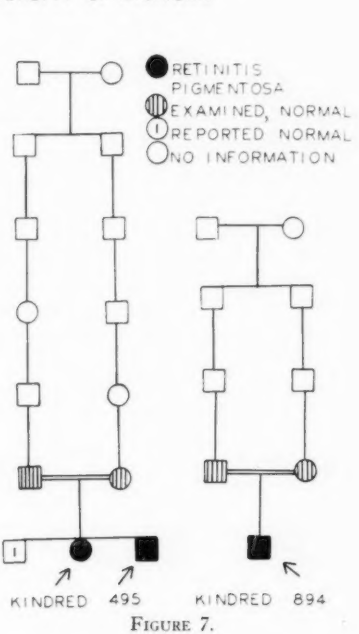


FIGURE 7.

In a high proportion of the pedigrees there can be demonstrated consanguinity of the parents.

3. Sex-linked recessive. This is the rarest form or pattern of hereditary transmission of retinitis pigmentosa. Three and possibly four such pedigrees are known.

4 and 5. Incomplete sex-linked. Incomplete sex-linked recessive and dominant modes of inheritance of retinitis pigmentosa have been postulated by J. B. S. Haldane.[5] While these views are as yet hypothetical, they must be presented for the sake of completeness. It is assumed that the genes are present in the homologous parts of the X and Y chromosomes. These pedigrees cannot be differentiated from the ordinary autosomal recessive or autosomal dominant pedigrees until they are pooled and analyzed statistically.

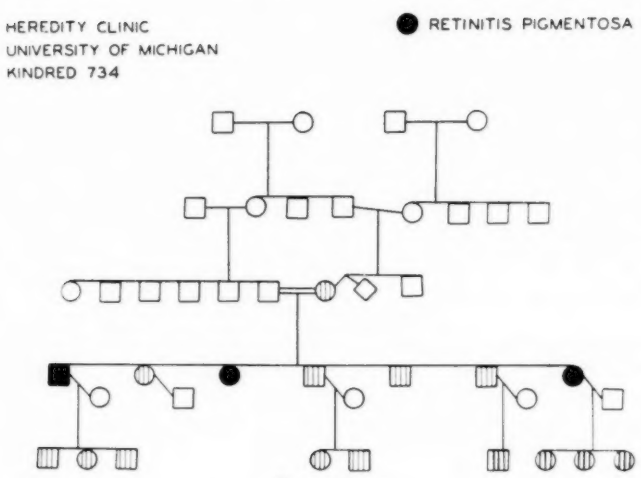


FIGURE 8.

In the dominant incomplete sex-linked form of pedigree it will be evident that if the abnormal gene is present on the Y-chromosome, it will appear generation after generation in all the males except for an occasional crossing over to the X-chromosome. When present on the X-chromosome, the retinitis pigmentosa will occur in equal sex proportion of a sibship when the parent is an affected female, but when the parent is an affected male, only the female offspring will be affected.

The incomplete sex-linked recessive form is without associated deafness. A recessive incomplete sex-linked gene reveals its presence by a disturbance of the expected 3:1 ratio among the offspring, both males and females. Thus a greater number of affected males or affected females in a sibship than the number anticipated would suggest the possibility of this mode of inheritance. In addition, there will be an unusually high incidence of consanguinity of the parents.

6. Intermediate sex-linked. Dr. Charles Cotterman and I[2] have recently reported a possible sixth mode of inheritance of primary retinal degeneration. This is an intermediate sex-linked X-borne trait. The males in the family reported demonstrated a severe progressive primary retinal degeneration, while the carrier or heterozygous female presented a mild and extremely slow specific aberration suggestive of gross irregularity of the surface of the lamina of Bruch — golden, glistening, minute drusen of widespread distribution being very characteristic of the latter.

Laurence-Moon-Biedl Syndrome (B-2-b, Leinfelder's classification). There are two chief theories as to the manner in which this syndrome is inherited: (1) that one gene (recessive autosomal) is responsible for the numerous defects, (2) that the changes are produced by two or more genes. The first theory in our opinion carries the least objection (fig. 9). Other syndromes exhibiting widespread ocular and constitutional polyphenous manifestations are known and are thought to be the effect of a single gene: Marfan's, the phakomatoses, and Bonnevie-Ulrich syndrome.

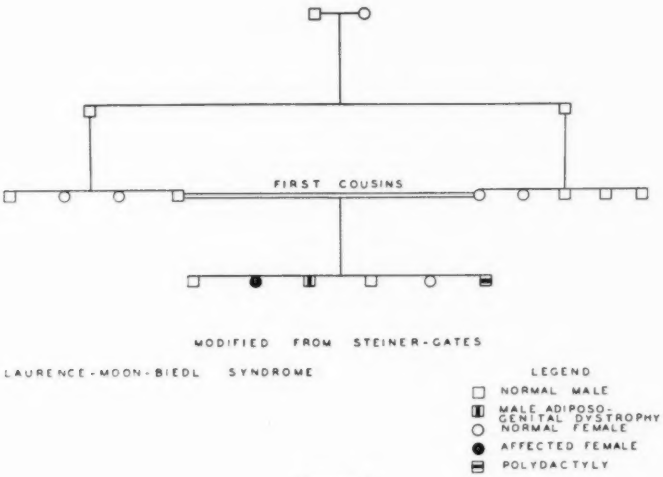


FIGURE 9.

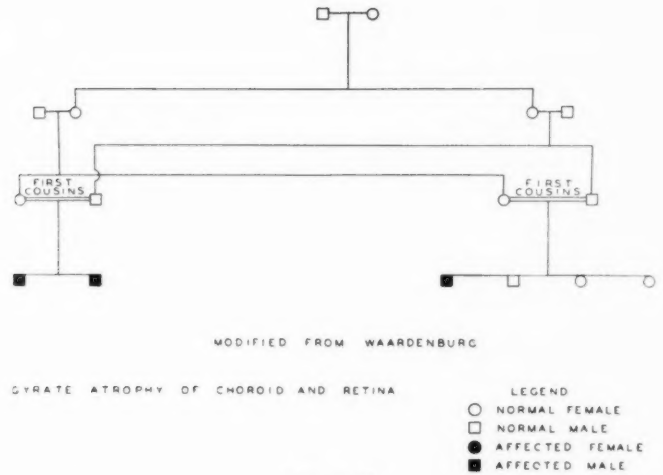


FIGURE 10.

C. *Progressive atrophy.*

1. *Gyrate atrophy of the choroid and retina* (fig. 10). The hereditary pattern is chiefly autosomal recessive, although there is some theoretic evidence that incomplete sex-linked recessive pedigrees may exist. Brothers and sisters of the same sibship may exhibit the disease. There is a high incidence of consanguinity among the parents. High myopia is a most frequent and common association.

2. *"Choroideremia" type.* We are, in a large measure, indebted to Drs. Clement McCulloch and R. J. P. McCulloch[6] for a rather complete explanation of the genetics of this disease (fig. 11). It is an intermediate sex-linked X-borne gene trait in which the heterozygous, or carrier, female exhibits a very specific but atypical picture of retinal and choroidal aberration but has no ocular symptoms. The males present a progressive choroidal abiotrophy leading to the typical and diagnostic retinal picture.

DISCUSSION

Allan[1] stressed that, in retinitis pigmentosa, certain specific clinical observations may be associated with different types of genetic behavior. He concluded that the disease was earlier in onset and more rapid in its course and that blindness ensued earliest when the genetic behavior was of the autosomal recessive type, that this was less true when the type was sex-linked recessive, and that they were even less marked in autosomal dominant. Allan emphasized that in the simple recessive form of the disease the gene was present in double dose and thus there was little or no amelioration effect from the other genes. In the sex-linked recessive mode of inheritance the abnormal gene is present on the X-chromosome and is therefore unopposed in the male, consequently it exerts a severe effect. The dominant autosomal gene, opposed as it is by the normal gene, usually presents a late onset and is

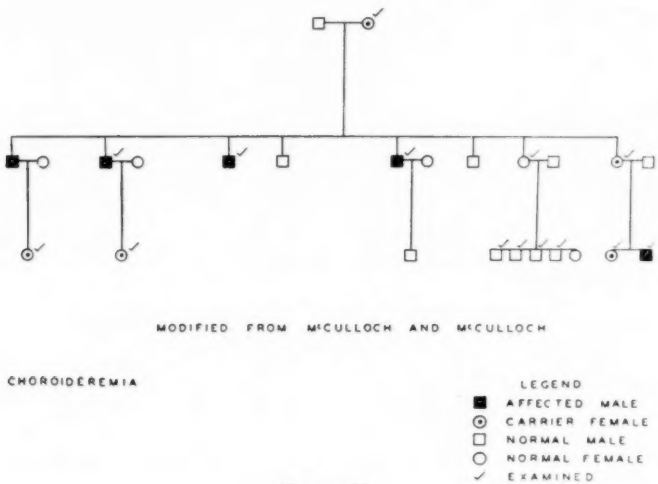


FIGURE 11.

Goedbloed[4] has recently confirmed this type of inheritance pattern. Dr. F. B. Fralick and I have three unrelated families which present the identical genotypical and phenotypical features of the disease.

A review of the literature indicates that the male picture is very similar. Heretofore the female carrier state has been missed or incorrectly labelled as "atypical retinitis pigmentosa."

mild in its course and influence on central visual acuity. This Allan attributed to the amelioration influence of the opposing normal gene. While one may generalize, as above, the observation does not entirely fit clinical observations. We have witnessed the onset of the rather typical autosomal recessive form of retinitis pigmentosa with deafness as late as the twenty-fifth year in specific families. Retinitis pigmentosa, as macular dystrophy, will vary tremendously between families as to onset, course and severity. There is, however, a great similarity within the specific family as to time of onset, course and severity of the disease. The disease will also exhibit moderate variation in clinical expression even between members of the same family. All that can be said is that there is a greater similarity between related afflicted individuals than exists between unrelated patients with the same affliction.

There has been and will be presented to you pathologic evidence of a wide variation in the degree of atrophic involvement of the rod and cone layer, the lamina of Bruch and the choriocapillaris. The physiologist considers these structures as a physiologic unit. In addition, there is some evidence that they may be considered as a genetic unit. The heterozygous carrier female in intermediate sex-linked inheritance in choroideremia, as in the family I have studied, presents what is interpreted as a mild gene effect interfering but little with the physiology of the eye but yet effecting alteration in the lamina of Bruch, in the retinal pigment and, questionably, in the choriocapillaris layer. The mild character of retinitis punctata albescens as to course and visual loss may be explained on a similar hypothesis, namely that of a mild chemical deficiency influencing all three layers but in varying intensities. In choroideremia the severe effect (male) is a total degeneration (abiotrophy) of the choroid. In gyrate atrophy it appears that there is also a slow secondary degeneration of the retina occurring concurrently with a patchy choroidal abiotrophy. The typical picture of retinitis pigmentosa has varied greatly; in one case there is seemingly a predominant primary degeneration of the rod (and cone) layer, but other cases exhibit a predominance of choroidal aberration. These variances in clinical and pathologic expression may have their counterpart in the great variation of the genetic patterns. The future holds the explanation! Let us hasten the day of understanding through careful study and analysis of all affected families by joint activity on the part of the ophthalmologist, the physiologist, the geneticist, the chemist and the internist.

CONCLUSIONS

I. Congenital Stationary Night Blindness
   1. Simple autosomal recessive
   2. Sex-linked recessive with myopia, nystagmus and occasionally fundus changes
   3. Simple autosomal dominant

II. Oguchi's Disease
   1. Simple autosomal recessive
   2. Theoretic incomplete sex-linked recessive

III. Retinitis Punctata Albescens
   1. Simple autosomal recessive

IV. Retinitis Pigmentosa
   1. Simple autosomal recessive
   2. Simple autosomal dominant
   3. Sex-linked recessive

In addition there is theoretical evidence that there exists

4. Incomplete sex-linked recessive
5. Incomplete sex-linked dominant

and a postulated

6. Intermediate sex-linked form

V. Laurence-Moon-Biedl Syndrome
   1. Simple autosomal recessive
   2. (?) Two recessive autosomal genes

VI. "Choroideremia" Type
   Intermediate sex-linked inheritance

VII. No eugenic advice should be given without a careful pedigree and meticulous examination of all of the family to furnish the necessary facts.

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PATHOLOGY

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THE pathologic aspects of primary retinal degeneration with night blindness revolve about retinitis pigmentosa, since specimens representing other clinical entities in this category have been too few to permit any comprehensive generalizations. There have, however, been an adequate number of authenticated cases of retinitis pigmentosa described, and these, together with some specimens not heretofore reported that have been made available to me, will comprise the substance of this report.* As part of the symposium, this study has the following aims: to describe the typical histopathologic characteristics of retinitis pigmentosa; to set forth the features enabling one to differentiate retinitis pigmentosa from conditions producing similar histologic changes; to discuss analogous though not necessarily similar histologic abnormalities of the retina; and to infer so far as possible the pathogenesis of retinitis pigmentosa and allied conditions.

In selecting cases on which a judgment is to be made of the typical pathologic changes of any disease entity, it is of course important that a correct diagnosis should have been made and that changes should not have occurred through other disease which might either mask or confuse the essential process under study. In the case of retinitis pigmentosa, these considerations are especially important, for although the clinical picture is highly characteristic, there are no histologic findings that are pathognomonic of retinitis pigmentosa; moreover, the occasion rarely arises when an eye with retinitis pigmentosa comes to enucleation without other disease of the eye. Accordingly, for the purposes of the present study only those cases are included in which there was adequate clinical evidence of retinitis pigmentosa prior to enucleation and in which there was either no other disease of the eye (postmortem specimens) or the other disease was considered to have produced no significant alteration of the retinal changes resulting from retinitis pigmentosa.

Five cases comprise the present study. Of these, 2 were removed post mortem, 1 was removed with absolute glaucoma, 1 was removed following an unsuccessful operation for glaucoma, and the other was removed for an acute inflammatory process in the anterior part of the eye. Of these, 2 were previously reported in the literature.[11,43]

The total number of pathologic descriptions recorded in the literature is about twenty. Of these, 9 cases may be considered authenticated by the clinical

From the Howe Laboratory of Ophthalmology, Harvard University Medical School, and the Massachusetts Eye and Ear Infirmary.
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*For the opportunity of studying these cases the author is indebted to the Army Institute of Pathology (Mrs. Helenor Wilder), the Johns Hopkins Hospital (Dr. Jonas Friedenwald), and the Verhoeff collection at the Massachusetts Eye and Ear Infirmary.

record, and the histologic changes due to retinitis pigmentosa were not obscured by other disease of the eye. These were the cases reported by Landolt,[23] Bürstenbinder,[8] Wagenmann,[46] Lister,[26] Ginsberg,[13] Suganuma,[39] Friedenwald,[11] Verhoeff[43] and the first case of the series of Koyanagi.[21] Many of the other cases frequently listed as retinitis pigmentosa were undoubtedly instances of the disease, but the absence of adequate clinical history or the presence of atypical features as cited below makes it seem unwise to include them in the present series.

Thus, Leber[24] reported that his patient was blind from birth. Poncet's case[30] was one of undoubted congenital stationary night blindness and not retinitis pigmentosa. Of Stein's 4 cases,[35] the first was syphilitic, the second and third had no clinical data reported, and although the fourth case had suggestive night blindness, there was also a history of interstitial keratitis suggestive of syphilis. Gonin[14] gave no history in his report, and the condition in his patient was noted to have been unilateral. Stock's cases[36,37] comprised a type of pigmentary degeneration beginning with loss of central field without night blindness leading to complete blindness within a few years and accompanied by degeneration in the central nervous system, which is unlike retinitis pigmentosa. Greeves' case[15] undoubtedly was retinitis pigmentosa, but enucleation had been preceded by two years of recurrent iridocyclitis, which may well have masked the histologic changes due to retinitis pigmentosa. Ascher's case[2] had insufficient clinical data to make a positive diagnosis of retinitis pigmentosa (the patient had died in the pre-Wassermann era), and there was the atypical (although very interesting) finding of a symmetrical fibroma-like mass of the choroid underlying the maculas of both eyes. Asayama and Takagi's patient[1] had advanced glaucoma and phthisis. Blum and Babel[6] gave data insufficient for a clinical diagnosis or for ruling out the presence of other ocular disease.

PATHOLOGY OF RETINITIS PIGMENTOSA

For the purposes of orientation it may be stated at the outset that the essential histologic features of retinitis pigmentosa consist in an absence of the rod and cone layer, an absence of most of the outer nuclear layer, an accumulation of melanin granules in the retina and varying amounts of gliosis. Noteworthy is the fact that the bipolar cell layer and the ganglion cell layer show little if any abnormality in uncomplicated cases of retinitis pigmentosa, and there is no optic atrophy.

The rod and cone layer had disappeared completely in all our cases except for the occasional retention of a few abnormal cones in the central area and in the far periphery (figs. 11, 21, 22). These cones were shorter, fatter and sparser than normal and simulated abortive cones occasionally seen in normal eyes adjacent to the disc and in the far retinal periphery. With the disappearance of the rods and cones, the external limiting membrane frequently became directly apposed to the pigment epithelium, but often it was separated from the pigment epithelium by a mixture of globular and amorphous debris (fig. 23). A frequent, although not constant finding, was the presence of a neuroglial membrane coursing horizontally just external to the outer limiting membrane and occupying the space of the rod and cone layer (figs. 1, 6, 12, 13). This membrane has been described by Verhoeff[43] and shown to be continuous with Müller's supporting fibers in the retina. It is made up of cells arranged more or less parallel to the outer surface, having nuclei that are larger in size and containing a greater abundance of protoplasm than do normal glial cells. So far

as could be determined in cross section, the external limiting membrane was usually preserved, although there is reason to believe that it may have become sievelike through the development of abnormal lacunas. An important feature of retinitis pigmentosa—and one which will be considered at greater length in discussing the differentiation from other conditions with pigmentation of the retina—is the absence of any real fusion of the retina to the outer layers (figs. 6, 8, 9, 13, 23). Except for a few occasional diaphanous strands in the far periphery (fig. 8), the retina in uncomplicated retinitis pigmentosa usually was not attached to the pigment epithelium, and in the preparation of histologic sections it separated from the outer layers just as does the normal retina (figs. 6, 8, 23). That separation of the retina may also occur during life in patients with retinitis pigmentosa is evident from the fact that three of the specimens with retinitis pigmentosa examined for the present study were excluded because of separated retinas.

The outer nuclear layer and even the outer plexiform layer were markedly attenuated or absent altogether, so that the bipolar layer came to lie directly against the external limiting membrane or was separated from it by delicate glial tissue only. This disappearance of the outer nuclear and outer plexiform layers was always more complete toward the periphery of the retina. Centrally there was usually preservation of a layer corresponding to the outer plexiform layer and frequently a solitary layer of neuronal cells lying on the external limiting membrane, presumably representing the nuclei of the retained cones.

The other layers of the retina (bipolar cell, inner plexiform, ganglion cell and nerve fiber layers) were normal (fig. 1) except for the presence of pigment and some gliosis, which in the case of the bipolar cell layer occasionally gave to it a moth-eaten appearance (fig. 6, 12). The inner layers of the retina were somewhat greater overlying the disc and occasionally showed a glial membrane extending into the vitreous (figs. 1, 12), but the gliosis of these inner layers was in no case conspicuously abnormal. It is true that some of the cases recorded as retinitis pigmentosa showed widespread gliosis and conversion of the retina into a glial scar, but all these cases showed other profound ocular disease, and the fact that other cases of retinitis pigmentosa, reported elsewhere and here, represented advanced instances of the disease without showing marked gliosis of the inner portions of the retina is evidence that this is not a characteristic of retinitis pigmentosa.

The retinal arteries showed moderate periarterial thickening and moderate hyalinization of their walls (figs. 5, 18), certainly much less than one would expect from the apparent constriction seen ophthalmoscopically in this disease.[25] Nor is it apparent why the retinal arteries *should* be narrowed, since the layers of the retina supplied by these arteries are so well preserved. The assumption that the constriction is an atrophy of disuse finds no histologic counterpart in retinitis pigmentosa.

It is generally stated that the vessels in the periphery of the retina show extensive hyaline occlusion, and since the hyalin is surrounded by a cuff of pigment cells, it has been suggested[23] that the occlusion has been brought about by the constricting effect of these latter cells. However, as will be pointed out subsequently, these hyalin-pigment collections are frequently found in the outer portion of the retina where there are no vessels normally, and the hyalin is more extensive than could be accounted for by vessel occlusion (figs. 10, 13, 15). It seems more reasonable, therefore, to consider this hyalin a product of the epithelium rather than an occluded ves-

sel. At least for the purpose of this review, no such area was considered a vessel unless red blood cells could be seen in it or unless an endothelial wall could be identified, and by these criteria there was not the extensive vascular change in the retinal periphery that has been previously reported.

The retinal veins frequently showed pigment in their outer walls, but it was also apparent that the pigment extended in branched fashion throughout the retina, sometimes connected with the pigment epithelium, not associated with the veins. While not denying that pigment ensheathing the veins accounts for much of the distribution of pigment in the retina, it seems likely that some of the angioid appearance of pigment strands is due to the insinuation of pigment epithelium in potential crevices in the retina unassociated with the veins.

The pigment granules in the retina appeared both free and intracellular, but in either case they often aggregated about blood vessels. There was nothing in the present study to indicate with certainty whether the cells containing the pigment were derived from the pigment epithelium and wandered into the retina, as frequently stated and illustrated so well in Ascher's article,[2] or were phagocytic cells[12,34] that had engulfed the pigment secondarily. Nevertheless, the cells were similar morphologically to those of the pigment epithelium; they had the same capacity to form clusters and secrete an eosinophilic hyaline material (figs. 10, 13, 15); the pigment granules in them had the same shape and the same distribution as in the pigment epithelium; and what was most impressive, they could occasionally be seen to be coming into the retina from the pigment epithelium (figs. 9, 16, 17). Thus the evidence suggests that at least some of the pigment-bearing cells within the retina are derived from the pigment epithelium. What the stimulus is for migration of pigment epithelial cells into the retina is, of course, subject to speculation. It is known, however, that there is a fundamental interdependence of the outer layer of the retina with the pigment epithelium. Thus, the pigment epithelial cells normally send delicate processes into the outer elements of the retina, forming intimate interdigitations with the rods and cones, and in some species at least, the cells have ameboid movements. Whether or not the processes are actively motile in human beings is not known, but the intimate connection between the outer layers of the retina and pigment epithelium is shown in human beings with separated retina; the epithelial cells in this condition proliferate and secrete a hyaline material to an unparalleled extent. In retinitis pigmentosa the external limiting membrane becomes directly apposed in many places to the pigment epithelium, and it has been suggested that the processes of these cells extend through the holes left in this membrane by the disappearance of the rods and cones,[10] and where the holes become big enough entire cells or groups of cells insinuate themselves through these holes. This migration of cells is not, then, fundamentally different from the normal capacity of the pigment epithelial cells to send processes into the crevices left by the rods and cones and may be a manifestation of a widespread phenomenon of ameboid cells such as is seen so beautifully in the case of neutrophile leucocytes which appear to "flow" through any abnormal holes in the blood vessel wall. At least this explanation, based on the assumption of an inherent ameboid property in the epithelial cells,[9] seems more palatable than the commonly stated ones that pigment is merely attracted forward into the retina by some heliotropic force[10] or that the epithelial cells wander into the retina in search of nutrition.[14]

Once in the retina the pigment-bearing cells do have a preference for the perivascular regions—this is especially well seen in flat sections of the retina—but they also form nests of cells independent of the vessels, as in the outer portions of the retina where there are no vessels (figs. 10, 14, 15, 21). Indeed, in cross sections of the retina one obtains the impression that the many pigment clusters which have their ophthalmoscopic counterpart in bone-corpuscle shapes owe their configuration to local proliferation of pigment cells and have no causal connection with blood vessels, as is generally assumed. In the retina the pigment cells may remain unchanged or may form a hyaline material similar to that of drusen of Bruch's membrane (figs. 10, 13, 15). In this latter case the pigment cells appear to surround the hyaline material (which often comes to look like an occluded vessel), and the hyaline material may become calcific centrally (fig. 19). On the other hand, the pigment cells may disintegrate, setting free pigment granules in the retina. Thus it is that some of the hyalocalcific areas come to be surrounded by a halo of scattered pigment.

It is worth pointing out that this description of pigment migration into the retina is not limited to retinitis pigmentosa. It is believed to occur in any pathologic process where the external limiting membrane comes in contact with the pigment epithelium. It occurs in chorioretinitis, degenerations of the macula, myopia and many other conditions. It is to be looked upon in retinitis pigmentosa, as in the other conditions, as a *secondary* process, usually if not always following degeneration of the rods and cones. This is mentioned here because the name retinitis pigmentosa and the outstanding clinical sign, the pigmentation, has led many to believe that the prime disturbance is linked with the pigment-bearing tissue.

As a membrane the pigment epithelium in retinitis pigmentosa is much more nearly normal than might be expected. There is some depigmentation (figs. 6, 7, 8, 12, 13, 23), especially in the peripapillary regions, and perhaps some attenuation of the cells, but the membrane is characteristically present as a monocellular membrane with no real hyperplasia or disintegration. The occasional occurrence of a hyaline excrescence was no more than might be expected in routine examination of eyes with no disease of the retina or choroid. The slight depigmentation and attenuation were thought to be consistent with the loss of epithelial cells that have been hypothecated to have migrated into the retina.

Bruch's membrane showed no significant abnormality (fig. 8), and the changes in the choriocapillaris were so slight as to be equivocal. There was some sclerosis of the choroidal arteries (figs. 6, 7, 23), perhaps somewhat more than might be expected on the basis of age but not more than is frequently found with other conditions unaccompanied by retinitis pigmentosa and, in any case, considerably less than the ophthalmoscopic appearances would lead one to expect. From the pathologic point of view, therefore, choroidal sclerosis may well be part of the picture of retinitis pigmentosa, but it was not sufficiently marked to be held accountable for the retinal changes. The choroidal veins and stroma showed no abnormality.

The optic nerve was essentially normal in 4 of the 5 cases examined by the present writer (the fifth case had absolute glaucoma), but optic atrophy is frequently described in the cases of the literature even when glaucoma is not present. The reason for this contradiction is not apparent. That optic atrophy is not to be passed off merely as a late change is evident especially from Verhoeff's case[43] in which the

patient was 60 at the time of the enucleation and had been blind from retinitis pigmentosa for twenty years. Drusen of the disc is an occasional finding ophthalmoscopically with retinitis pigmentosa, and one of the cases here reported (the one with advanced glaucoma) had such a finding histologically (fig. 20). It consisted of a calcific mass occupying most of the substance of the nerve head anterior to the lamina cribrosa.

Gliosis of the optic nerve was an inconspicuous feature in the cases studied except where glaucoma was present and was much less than is frequently found in routine histologic specimens with a variety of conditions. There was found little on histologic study to account for the waxy color which has been so often described ophthalmoscopically and which has been attributed to gliosis of the disc. A possible explanation is, of course, that some gliosis of the disc, however mild, in the presence of a normal optic nerve may produce a discoloration which might not be apparent in the presence of an optic atrophy.

PSEUDORETINITIS PIGMENTOSA

The name "pseudoretinitis pigmentosa" is intended to refer to cases in which the histologic picture has many of the features characteristic of retinitis pigmentosa but in which the weight of evidence indicates that the process results from some disease other than the abiotrophy known as retinitis pigmentosa.* The name "pseudoretinitis pigmentosa" is not ideal, but it is considered less objectionable than such other terms as "secondary pigmentary degeneration of the retina," since, as already stated, the pigmentation of the retina in retinitis pigmentosa is also a secondary and incidental manifestation. The name "pseudorentitis pigmentosa" also has the advantage of emphasizing what from the pathologic point of view is its most important feature, namely, the confusion which it gives rise to in regard to retinitis pigmentosa.

Pseudoretinitis pigmentosa may be the result of any of a number of disease processes. The cases considered in the present study were instances of long-standing dislocation of lens into the vitreous, trauma in childhood, especially that resulting in staphyloma of the cornea, and corneal ulcer in childhood with perforation and secondary glaucoma.* Whether or not an inflammatory process had been present in all cases could not be determined, but it is noteworthy that an inflammatory episode, if it had existed, was not known to the patient in many of the cases. It is of interest that none of the cases gave a history of primary uveitis and none of syphilis, although syphilis is known to produce a similar histologic picture.[35] That glaucoma alone cannot produce this picture is suggested, however, by the absence of any comparable histologic findings with buphthalmos. In all cases the eyes were completely blind and had been so for a long time (years) prior to the enucleation. The reason for the enucleation was either pain from glaucoma or confusion with tumor.

In common with retinitis pigmentosa, pseudoretinitis pigmentosa shows disappearance of the rod and cone layer (figs. 24, 25, 26, 27, 30, 31, 32), replacement of the outer nuclear layer by neuroglial tissue (figs. 24, 25, 30, 35), migration of pigment cells into the retina

*The name pseudoretinitis pigmentosa has previously been used in a histologic sense, as intended here, by Blum and Babel (6) and also as a clinical term referring to the same group of cases by Fuchs (Atlas of Histopathology of the Eye, Leipzig, Deuticke, 1924).

*The relation of what we call pseudoretinitis pigmentosa to staphyloma of the cornea and secondary glaucoma was noted by Fuchs (Atlas of Histopathology of the Eye, Leipzig, Deuticke, 1924, Plate 12, fig. 3) and was thought to be due to inflammatory process in the retina consequent to the affection of the anterior part of the eyeball or to a shifting of the retina over the choroid (Fuchs, Ernest: Uber Verziehung der Netzhaut und Papille, Arch. f. Ophth. 104:230-246, 1921).

(fig. 29), especially about blood vessels, and subsequent changes in these cells as previously described (figs. 24, 25, 26, 27, 28, 33). Isolated fields of the one condition may be indistinguishable from the other (figs. 24, 25, 31). Yet the over-all picture is different, and this differentiation is most apparent with the low power of the microscope. The inner layers of the retina with pseudoretinitis pigmentosa show much more disorganization and gliosis than is present in uncomplicated cases of retinitis pigmentosa (figs. 26, 34, 35); the pigmentation of the retina is apt to be much denser and more marked in pseudoretinitis; there appears to be a greater number of degenerative products (hyaline and calcific bodies), although this is not constant; and what is most important, there is more extensive, albeit focal, disappearance of pigment epithelium and intermittent fusion of the retina with the chorioid (figs. 26, 30, 32, 34, 35). This fusion of the retina with the outer layers is rarely if ever more than a tenuous affair in the case of retinitis pigmentosa, and then the retina is attached by strands to the pigment epithelium, whereas in pseudoretinitis pigmentosa these are firm unions of the retina with the inner layers of the choroid. Thus the retina in retinitis pigmentosa separates readily in preparing the histologic preparations, just as does the normal retina, whereas it does not do so in pseudoretinitis pigmentosa. A further sign of differentiation, although not always present, is the scarring of the choroid in the case of pseudoretinitis pigmentosa, usually most evident in the regions of chorioretinal fusion (figs. 26, 27). A notable exception to this is the case, presumably of retinitis pigmentosa, reported by Ascher[2] in which there was extensive fibrosis of the choroid in the macular regions; nevertheless, even in this case there was no disappearance of the pigment epithelium and no chorioretinal fusion. The pigment epithelium is more profoundly altered in pseudoretinitis pigmentosa than is the case in true retinitis pigmentosa. Whereas in the latter condition it is usually preserved as a continuous monocellular membrane, in the former condition it is absent over considerable stretches and, where present, may be proliferated to form irregular thicknesses several cells deep (figs. 26, 30, 32, 34, 35). Yet isolated areas may show it to be intact, appearing to be made up of normal or depigmented cells as in the case of retinitis pigmentosa (fig. 31).

One case falling into this category which might be termed "focal pseudoretinitis pigmentosa" showed areas of absence of the rods and cones and of the outer nuclear layer over short stretches only, the retina elsewhere being normal (fig. 36).

OTHER CONDITIONS HAVING HISTOLOGIC CHANGES SIMILAR TO RETINITIS PIGMENTOSA

There are a number of conditions which are symptomatically similar to retinitis pigmentosa and might be expected to show similar lesions of the retina. These are congenital stationary night blindness, retinitis punctata albescens, gyrate retinal atrophy, Oguchi's disease, choroideremia and chronic vitamin A deficiency. Specimens have been examined in some of these. There is also a variety of experimental and idiopathic conditions in animals which have been found to have similar histologic changes.

It is generally stated that no anatomic studies have been made in cases of congenital stationary night blindness.[4] It should be noted, however, that the case reported histologically by Poncet[30] and clinically by Hocquard[16] as retinitis pigmentosa and usually included under this heading in the subsequent literature was undoubtedly congenital stationary night blindness. Unfortunately, the findings in this case, the only one of its kind studied

histologically, were inconclusive as regards the rods and cones because of the extensive postmortem changes, but it was noted that all the nuclear layers of the retina were normal.

The one instance of Oguchi's disease which was studied histologically[29] was noted to have shown an anomalous distribution of cone cells, many cone nuclei placed posterior to the external limiting membrane (in the "vorgelagerten" position), and a reduplication of the pigment epithelium, but the changes are not dissimilar to those frequently found in a variety of normal and pathologic specimens, and it seems entirely proper to question their significance[42] in the case reported as Oguchi's disease.

Several cases of the condition listed as choroideremia have been reported by McCulloch.[27] Whether or not they are accepted as genuine cases of choroideremia depends on one's definition of the term. The patients in question had a progressive disease characterized subjectively by night blindness and objectively by degeneration of retina and loss of choroid and pigment epithelium. It was manifest as such only in the males, while the females had no subjective symptoms but objectively showed a nonprogressive, granular type of pigmentation. The specimens which the authors kindly permitted me to examine showed the following changes (figs. 37 and 38). The outer elements of the retina were missing except for occasional abortive elements. For the most part, however, the rod and cone layer was missing completely or replaced by neuroglial tissue. The outer nuclear layer was similarly missing, as might be expected. The bipolar cell layer was also markedly attenuated. The ganglion cell layer, however, seemed normal, at least about the disc. There was moderate and variable thinning and gliosis of the retina. In places the retina was reduced to a thin glial membrane having none of the landmarks of the normal retina. No pigment was seen in the retina. The pigment epithelium was present in isolated areas only, being characteristically absent from most of the back of the eye. The choroid was variable. It was almost completely absent over about one-third of the posterior segment, and here the retina, or what remained of it, was represented by a few connective tissue strands. Elsewhere the choroid was present but characterized by marked sclerosis of all the choroidal vessels. There was extensive fusion of the retina to the choroid so that artefact separation of the retina usually took the choroid with it. In comparison with the histologic changes in retinitis pigmentosa these cases similarly showed degeneration of the outer retinal elements, but there was more disorganization of the retina, fusion of the choroid to the retina, and extensive atrophy of the choroid. It is not impossible that the extensive sclerosis of the choroidal arteries with consequent ischemia may in these cases alter the primary changes. At least in these cases the final histologic picture is more like that which has been described following occlusion of the blood supply to the choroid rather than the picture which has been described as typical of retinitis pigmentosa. It also should be noted that gliosis of the retina may conceivably be so marked in cases of retinitis pigmentosa as to simulate choroideremia by causing a diffuse white opacity that hides the choroid.[44] It is also not impossible that extensive fibrosis of the choroid, such as occurred in Ascher's case[2] with elimination of all but the larger choroidal vessels, or the massive metaplasia of the pigment epithelium that was referred to by Verhoeff[45] would produce an ophthalmoscopic picture that simulated choroideremia, although it would have to be much more widespread than was the case with either Ascher's or Verhoeff's case.

Retinal changes from primary vitamin A deficiency have not been described in human beings so far as I am aware, but the similarity of the changes observed in chronic experimental avitaminosis A and those of retinitis pigmentosa are so striking as to merit consideration here. These changes as observed in rats and dogs[18,40,41] consist in degeneration of the rods and cones and of the outer nuclear layer of the retina with preservation of the bipolar-cell and ganglion-cell layers. In other words, chronic avitaminosis A, which is known to produce the same functional abnormality as is found in retinitis pigmentosa, produces basically the same histologic changes in the retina. There have been several studies made on eyes removed from patients with night blindness and icterus from liver disease, but histologic changes in the retinas of these cases were either absent[3,17,21] or indistinguishable from postmortem artefacts.[31]

Worthy of passing note are lesions selective of the outer layer of the retina in experimental poisoning with certain iodate compounds[19] and adrenalin compounds[33] and the idiopathic occurrence of such lesions in certain strains of rats and mice either as a progressive[7] or stationary[20] process.

Lesions of the posterior ciliary arteries also result in degeneration of the outer portions of the retina[5,9,22,47] which have been likened to retinitis pigmentosa but it would appear that the degeneration in this experimental ligation of the ciliary arteries is more profound than occurs in retinitis pigmentosa of human beings, perhaps more analogous to the changes occurring as choroideremia or to the changes occurring following opticociliary neurectomy in human beings (fig. 39).[10,38]

It was at one time thought that the pathologic events in retinitis pigmentosa might be attributable to impairment of the choroidal circulation but this has not been borne out by morphologic changes in the choriocapillaris of the cases of retinitis pigmentosa which have been studied histologically.[28] Nevertheless, it cannot be denied that there are many instances in human ocular pathology in which choroidal ischemia leads to degeneration of the outer retinal elements. This is perhaps most strikingly exemplified in cases of melanoma of the choroid. Benign melanomas characteristically spare the choriocapillaris and are not accompanied by any degeneration of the overlying rods and cones, while the malignant melanomas replace the choriocapillaris over variable extents and are accompanied by corresponding disappearance of the outer retinal elements.

CONDITIONS HAVING HISTOLOGIC PICTURES ANALOGOUS BUT NOT SIMILAR TO RETINITIS PIGMENTOSA

In a broad sense any condition in which there is a selective disappearance of the percipient retinal elements and their nuclei with preservation of the rest of the retinal elements is analogous to retinitis pigmentosa. While this occurs in a variety of conditions, its limitation to a local area offers a means by which the over-all picture may be readily differentiated from retinitis pigmentosa. Nevertheless, study of isolated microscopic fields may be confusing, and on this basis there may be some merit in referring to disappearance of the outer retinal elements as analogous pathologic change. Indeed, some of the conditions may be looked upon as a localized retinitis pigmentosa.

First of all, it is important to remember that the most peripheral portions of the retina frequently show an absence of the outer elements of the retina, tenuous connections with the pigment epithelium, attenuation of pigment in the epithelium, and frequently migration of pigment into the retina as in retinitis pigmentosa. This may extend 2 to 3 mm.

from the ora and, although occurring most frequently in myopic eyes, is so common as to be considered a normal variation.

The macular changes with some of the hereditary dystrophies (cerebromacular degenerations, type Vogt-Spielmeyer) are analogous to retinitis pigmentosa, although involving preferentially the central rather than the peripheral retina. Indeed they have been called, on this basis, "retinitis pigmentosa inversa."[32] Pathologically there is a selective disappearance of the outer retinal elements about the macula and local migration of pigment into the retina. The overlying pigment epithelium is attenuated and depigmented, but the choroid is relatively normal.

Senile macular degeneration is also analogous in that there is preferential degeneration of the outer retinal elements and migration of pigment into the retina, but there is more extensive involvement of all the retinal layers, more evidence of vascular disease in the choroid, proliferation of the pigment epithelium and genuine fusion of the choroid to the retina (figs. 40 and 41). Accordingly, it is more analogous to pseudoretinitis pigmentosa than to retinitis pigmentosa proper.

SUMMARY, CONCLUSIONS AND SPECULATIONS

The prime pathologic changes in retinitis pigmentosa consist of disappearance of the rods and cones and outer nuclear layer. The inner retinal layers are characteristically preserved even late in the disease. The pigmentation of the retina is a secondary process, probably conditioned by having the sievelike external limiting membrane in contact with the pigment epithelium with subsequent migration of epithelial cells into the retina and leading to diminution in the pigment content of, and some attenuation of, the remaining pigment epithelium. The migrated pigment epithelial cells undergo a number of proliferative and degenerative changes in the retina. The histologic picture of retinitis pigmentosa differs from the nonabiotrophic type, here called pseudoretinitis pigmentosa, in showing less extensive disruption of the inner retinal layers, less pigmentation and less fusion with the choroid.

The prime histologic changes of retinitis pigmentosa are basically similar to those occurring with experimental chronic vitamin A deficiency, some types of poisoning and with certain idiopathic abnormalities in animals. With choroideremia (McCulloch) and interruption of the circulation to the choroid, the retinal changes are so marked as to prevent determination of what the prime changes were. In certain hereditary and senile degenerations of the macula, disappearance of the outer retinal elements with migration of pigment into the retina occurs in a manner analogous to what takes place in retinitis pigmentosa.

Although the morphologic changes in the retina suggest the primary lesion is in the percipient elements and in their nuclei, it is by no means excluded that the fundamental abnormality is a metabolic one in which degeneration of the retinal elements is secondary. It is not impossible, for instance, that the prime abnormality is in a local inability to utilize the vitamin A supplied to the pigment epithelium. It would seem reasonable at the present time to suppose that owing to some defect in the enzyme system the rods and cones or the pigment epithelium were unable to utilize the vitamin A supplied to them. Such a hypothesis seems the most reasonable explanation for the similarity of the pathologic changes in retinitis pigmentosa of human beings to those of chronic vitamin A deficiency in animals.

PROTOCOLS

Case 1

Retinitis pigmentosa. Reported by Frederick H. Verhoeff: Arch. Ophth. 5:392, 1931. The specimens of this case were perhaps the most satisfactory to study, since the eye had been enucleated during life and, having been fixed in Zenker's solution, had permitted differential staining for neuroglia with phosphotungstic acid (figs. 1 through 5).

Case 2

Retinitis pigmentosa. Reported by Jonas S. Friedenwald: Arch. Ophth. 4:767, 1930. The patient had syphilis, but the history of onset and the ophthalmoscopic findings were typical of retinitis pigmentosa.

It is perhaps worth drawing attention to the fact that the patient was a Negress, since it is often stated (although I cannot find any authoritative source for it) that retinitis pigmentosa does not occur in the colored race.

Case 3

Retinitis pigmentosa. Specimen submitted to the Army Institute of Pathology by Dr. Blankstein. AIP 100945. The patient was a 46-year-old woman who had typical ophthalmoscopic findings of retinitis pigmentosa and myopia. The eye was removed following an unsuccessful operation for glaucoma. The blood Wassermann test was negative. Other members of the family had typical retinitis pigmentosa.

The specimens consisted of cross sections of an eye of approximately normal dimensions (figs. 6 through 10).

The anterior segment of the eye showed the evidence of glaucoma (peripheral anterior synechia), bullous keratopathy, and rupture of lens with incarceration of lens capsule in corneal wound.

The posterior segment of the eye showed the following abnormalities in the retina. There was complete absence of the rods and cones and replacement of the outer nuclear layer by neuroglial tissue. The external limiting membrane was intact but showed occasional holes of the order of magnitude of 0.1 mm., which appeared filled by pigment cells. The outer portions of the retina were separated from the pigment epithelium (by artefact) except in the most anterior regions, where there were tenuous strands connecting the retina and pigment epithelium but no real fusion. The regions containing these tenuous strands appeared to be places where the pigment epithelium had proliferated into the retina, since there were pigment cells in the corresponding portions of the overlying retina and relative depigmentation of the underlying pigment epithelium. The pigment epithelium was everywhere present as a monocellular membrane

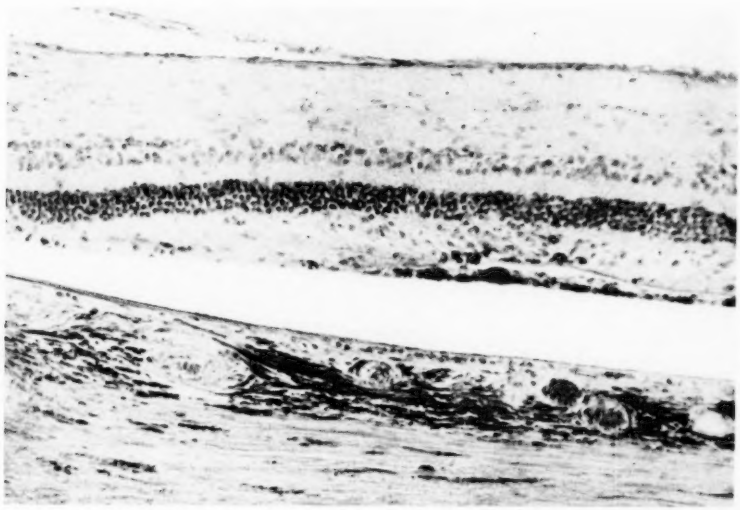

FIG. 1—Case 1. Low-power view of retina and choroid in region of disc showing glial membrane on inner surface of retina, preservation of ganglion-cell and bipolar-cell layer, and replacement of outer retinal layer by neuroglial tissue.

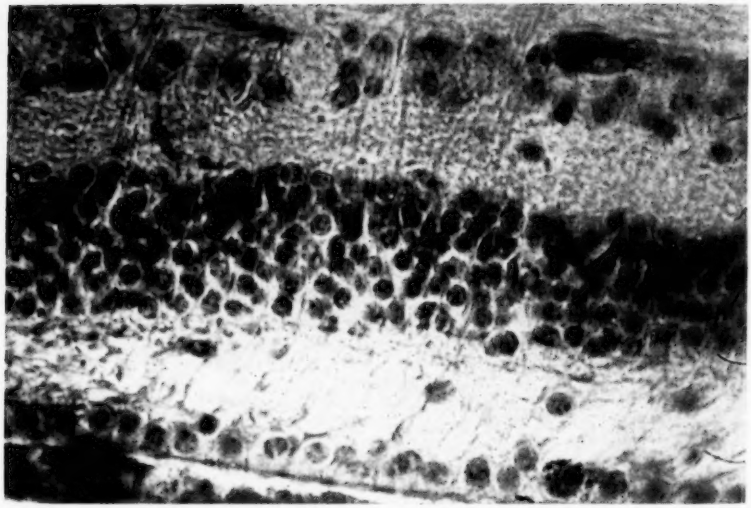

FIG. 2—Case 1. High-power view of retina and pigment epithelium in region of fovea showing preservation of a single row of nuclei of the outer nuclear layer (presumably cone nuclei).

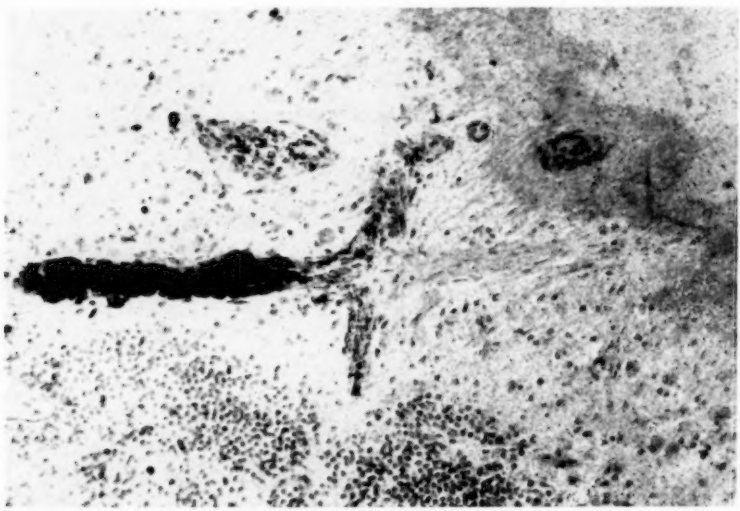

FIG. 3—Case 1. Low-power view of retina cut on the flat showing pigmented cells in clusters and in dendriform shapes spreading throughout the retina. While some of the configurations appeared to follow the course of vessels, there was no recognizable vessel wall. It is suggested, therefore, that the vessel-like distribution is due to the insinuation of proliferating pigment epithelium into potential crevices of the retina.

FIG. 4—Case 1. High-power view of area shown in figure 3 showing rarefaction and absence of pigment in many of the cells thought to have been derived from the pigment epithelium.

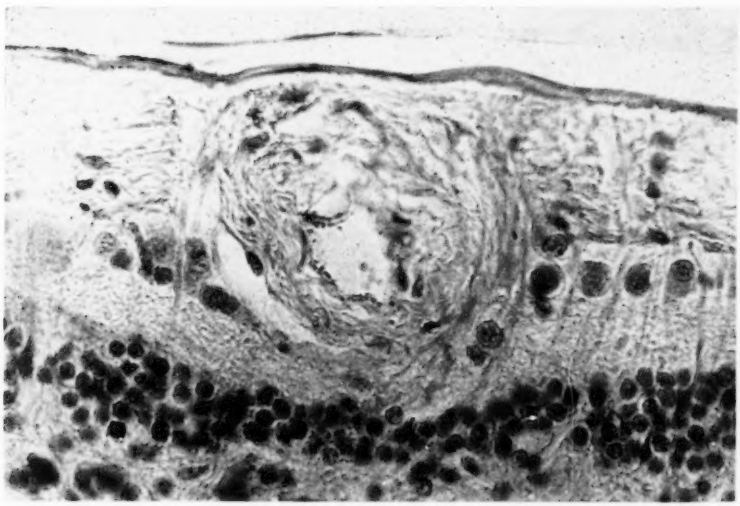

FIG. 5—Case 1. Cross-section of retinal artery at the inner portion of the retina showing considerable thickening of the arterial wall and of the inner limiting membrane of the retina. Such marked changes in the vessel walls were unusual in the cases of retinitis pigmentosa studied.

and appeared normal except for the local depigmentation anteriorly in connection with the tenuous strands and a generalized depigmentation posteriorly in the central and peripapillary regions.

The bipolar layer of the retina was present throughout but did have a slightly moth-eaten appearance, especially in its outer aspects where neuroglial tissue encroached on it. The inner layers of the retina showed some ganglion cells, but whether or not these were diminished in number could not be ascertained, since the sections studied did not go through the macula. There was no appreciable gliosis of the inner layers of the retina.

The optic nerve showed mild atrophy with some gliosis of its prelaminar as well as its postlaminar portions. No pathologic cupping was evident.

The choroid was normal except for moderate sclerosis of the choroidal arteries; the choriocapillaris was normal.

Case 4

Retinitis pigmentosa. Case submitted to the Army Institute of Pathology by Dr. Ellett AIP 81319. The patient was a 26-year-old woman who had been observed since the age of 6 and clinically had typical retinitis pigmentosa. One sister was known to have retinitis pigmentosa. The patient's vision is said to have failed gradually, and at the time of enucleation she had light perception only. About four months prior to enucleation, the pupil was artificially dilated, and the patient subsequently developed severe pain (presumably glaucomatous). Iridectomy was done eight days prior to enucleation, but hemorrhage into the anterior chamber occurred on the third postoperative day, and the eye was removed.

The specimens consisted of cross-sections of an unusually small eye (measuring 20 by 20 mm.).

The anterior segment showed peripheral anterior synechia (about 0.3 mm. in length), unusual amount of pigment in the angle, blood in the anterior chamber, and early necrosis of the iris.

The posterior segment showed the following changes in the retina. The rods and cones were completely absent except for a peripheral zone extending from the ora serrata posteriorward for a distance of approximately 2 mm., where there were definite, although stunted, cones (fig. 11). The outer retinal layers were everywhere replaced by neuroglial tissue except for the aforementioned area just behind the ora serrata where the

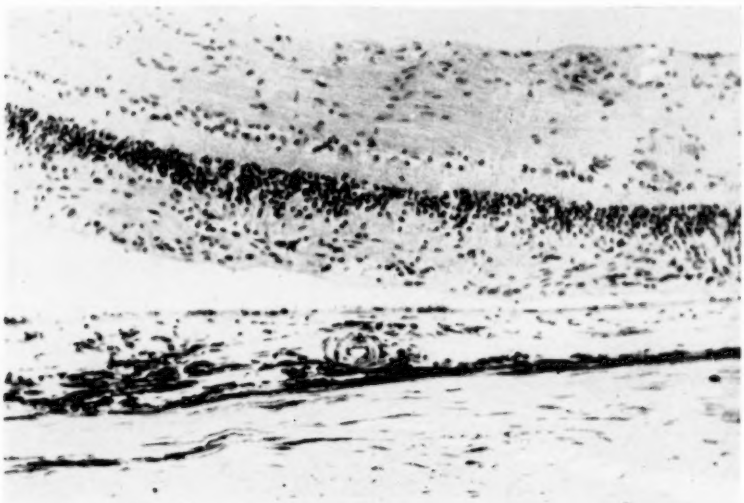

FIG. 6—Case 3. Low-power view of retina and choroid in posterior portion of globe showing moth-eaten appearance of bipolar-cell layer, replacement of outer layers by neuroglia, absence of fusion of retina with outer layer, relative depigmentation of pigment epithelium and sclerosis of choroidal artery.

FIG. 7—Case 3. High-power view of same field showing depigmentation of pigment epithelium and sclerosis of choroidal artery.

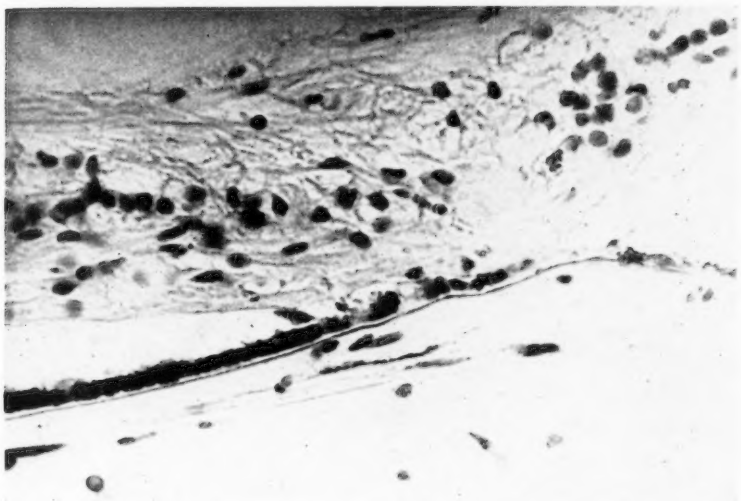

FIG. 8—Case 3. High-power view of retina and pigment epithelium in anterior aspect of globe showing tenuous connection of retina with outer layers and local depigmentation of the pigment epithelium but no real fusion. Pigment granules may be seen in the retina overlying the area of tenuous connection. Bruch's membrane may be seen to be intact beneath the pigment epithelium.

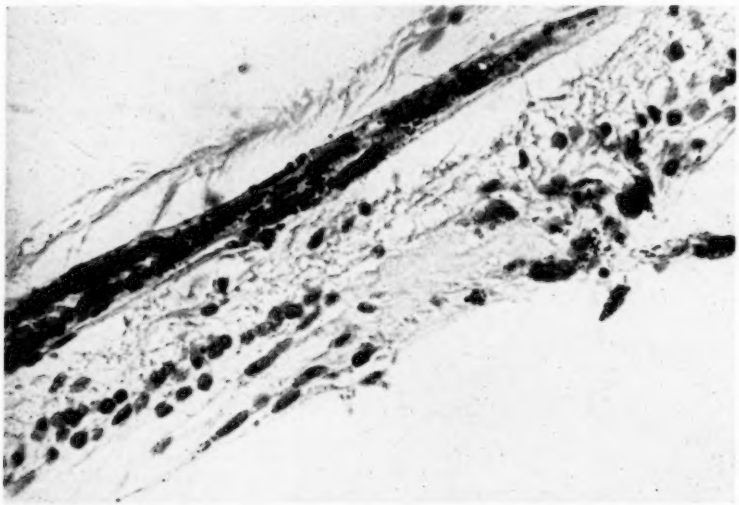

FIG. 9—Case 3. High-power view of retina in the equatorial zone showing a linear arrangement of pigment cells in the inner portions of the retina and a small portion of the pigment epithelial layer that adhered to the outer portion of the retina when the latter was artefactually separated. Although the line of pigment cells might suggest a relationship to a blood vessel, no evidence of the vessel was to be found in the sections. The small segment of pigment epithelium adherent to the outer portions of the retina (with some pigment in the overlying portions of the retina) is interpreted as illustrating the migration of pigment epithelium into the retina.

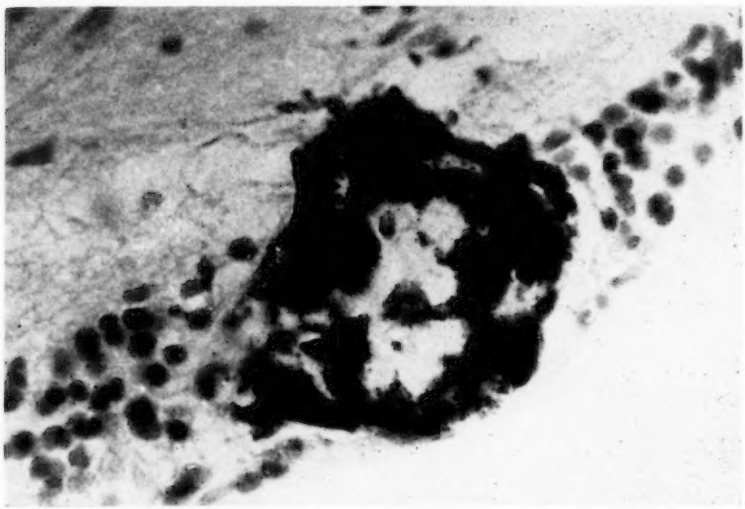

FIG. 10—Case 3. High-power view of pigment cluster in outer portion of retina with hyaline center. Such appearances have previously been interpreted as pigment about a hyalinized vessel, but since no sizeable vessels are situated in the outer retina, and since various stages in the formation of such a cluster can be traced from the pigment epithelium, it seems more likely that such a formation is due to the proliferation of pigment epithelial cells in the retina and secretion by them of a hyaline substance. This latter is perhaps an aberrant attempt to form Bruch's membrane.

outer layers were approximately normal (figs. 12 and 13). Much of the neuroglial tissue was vacuolated, appearing in low-power magnification like myxomatous tissue. The external limiting membrane was recognizable over much of the outer portions of the retina but showed many ruptures filled with pigment cells. Some of these pigment cells were arranged in free clusters, but many were still connected with the pigment epithelium (figs. 14 and 15). The pigment epithelium was practically everywhere intact and mostly present as a monocellular membrane, but in places it was profoundly altered. There were numerous focal areas of proliferation with extensions into the retina and, rarely, a drusen formation. Whereas the portion of the pigment epithelium in the equatorial or pre-equatorial part of the globe showed the normal amount of pigment, that in the posterior part showed such marked depigmentation that with the low-power lens it was often difficult to identify the epithelium between the retina and choroid.

The bipolar cell layer was recognizable but considerably distorted by the neuroglial tissue. The ganglion cell layer showed considerable increase in neuroglial tissue with only a rare ganglion cell visible. There was considerable pigment in the retina, both about blood vessels and in clusters not apparently connected with blood vessels (e.g., in the outer layers of the retina). The blood vessels showed slight hyaline and cellular thickening of their walls but were not greatly abnormal (figs. 16, and 17, 18, 19).

The optic nerve was markedly atrophic and showed considerable gliosis of its prelaminar and postlaminar portions but not more than is frequently seen with glaucoma. The lamina cribrosa showed considerable bowing backward. The most noteworthy abnormality in the optic nerve was a calcific body measuring approximately 0.5 mm. in diameter and replacing much of the substance of the nerve head in front of the lamina cribrosa (fig. 20). This is undoubtedly a drusen of the optic nerve head.

The choroid showed extensive sclerosis of the arteries, and the choriocapillaris was considerably attenuated. The entire choroid was thinned and somewhat fibrotic.

Comment: Although the patient had advanced glaucoma (as evidenced by the anterior synechia and degeneration of the ganglion cells), most of the retinal changes were similar to those in the other cases of retinitis pigmentosa. There was, however, more gliosis of the retina and a greater tendency to fusion of the retina to the choroid. The sclerosis of the choroidal arteries was also more marked, and an especially noteworthy finding was the

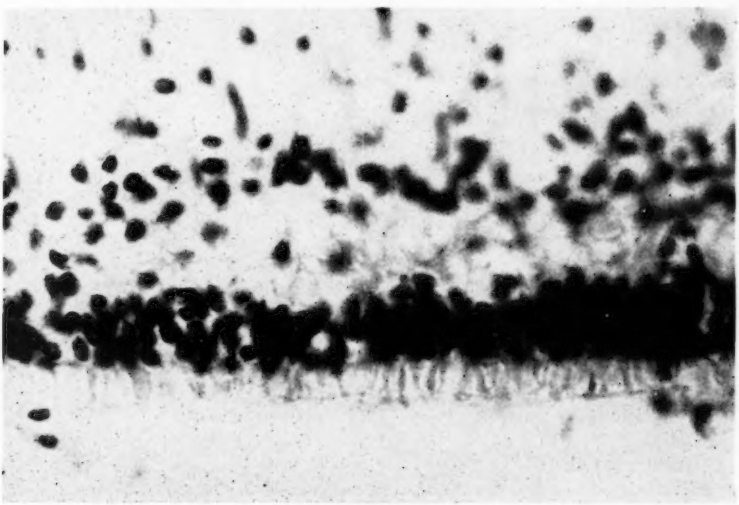

FIG. 11—Case 4. Outer portion of peripheral retina showing preservation of outer nuclear layer and stunted cones.

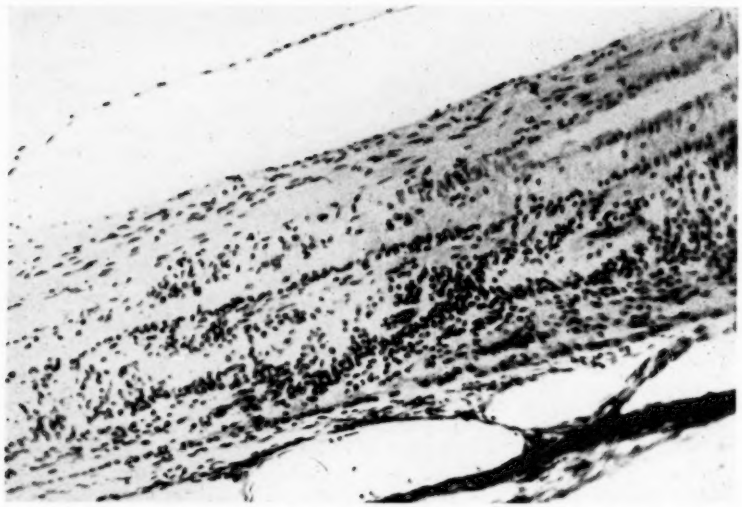

FIG. 12—Case 4. Retina and choroid in region adjacent to disc showing glial membrane extending into vitreous, considerable distortion of the bipolar-cell layer, replacement of the outer layers by neuroglia, and marked depigmentation of the pigment epithelium.

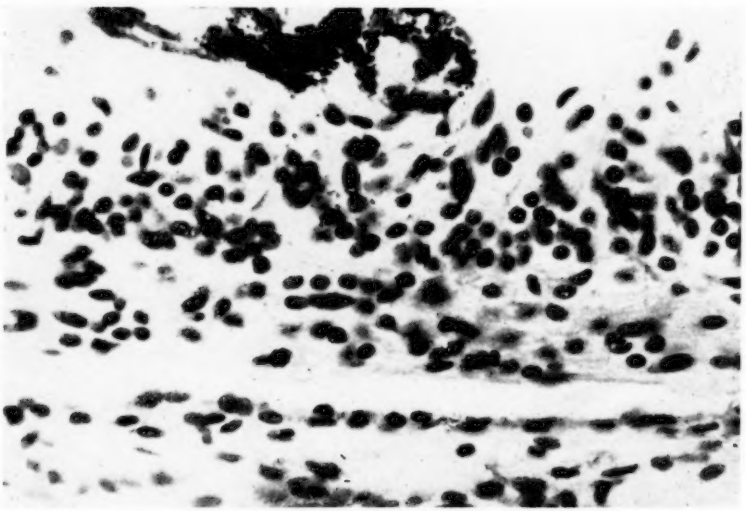

FIG. 13—Case 4. High-power view of outer portion of the retina showing a pigment cluster in the retina, replacement of the outer layers by neuroglia, absence of any real fusion, and depigmentation of the pigment epithelium.

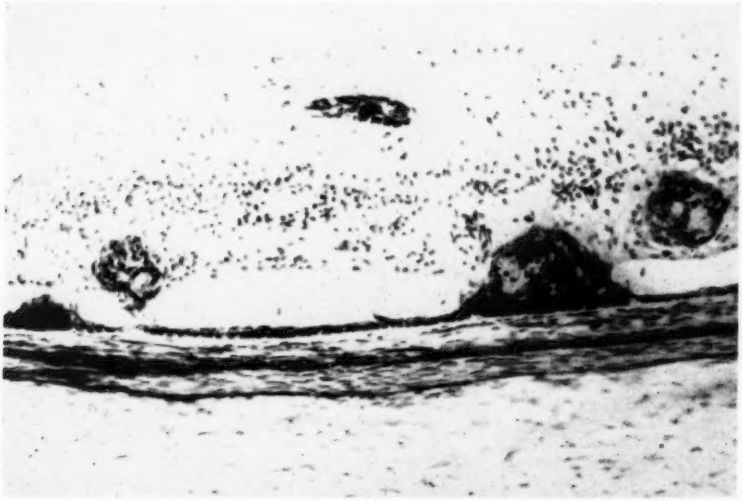

FIG. 14—Case 4. Low-power view of retina and choroid showing focal proliferation of pigment epithelium and free pigment clusters within the retina.

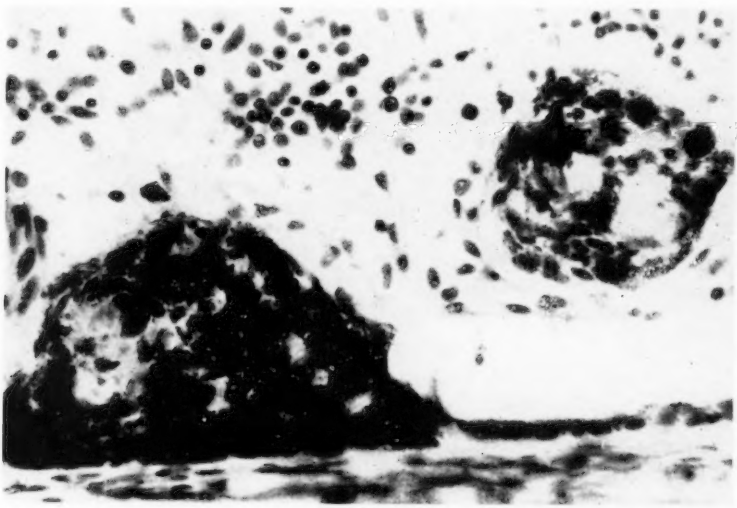

FIG. 15—Case 4. High-power view of same field showing one cluster still attached to the epithelium and another in the outer portion of the retina having a hyaline center simulating occluded blood vessels.

FIG. 16—Case 4. Low-power view of retina showing proliferation of pigment epithelium into region of a blood vessel in inner layers of retina.

FIG. 17—Case 4. High-power view of same field showing relationship to the blood vessel.

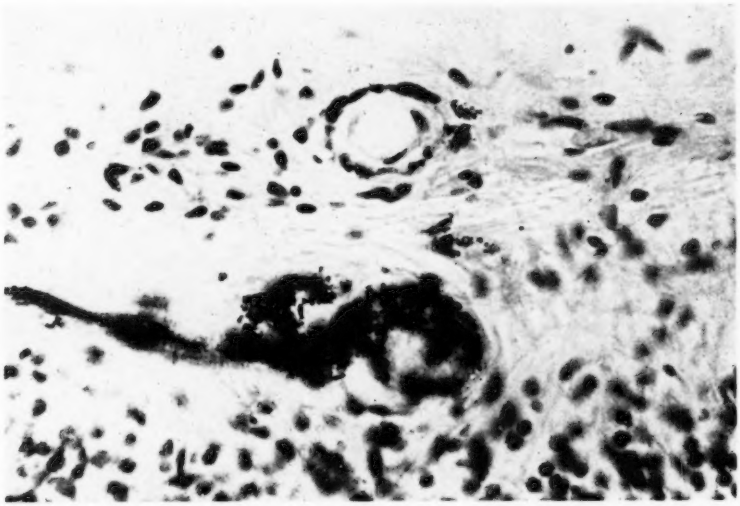

FIG. 18—Case 4. High-power view of inner retinal layers showing a pigment cluster and a vessel with slightly hyaline wall and a small amount of pigment in the vicinity.

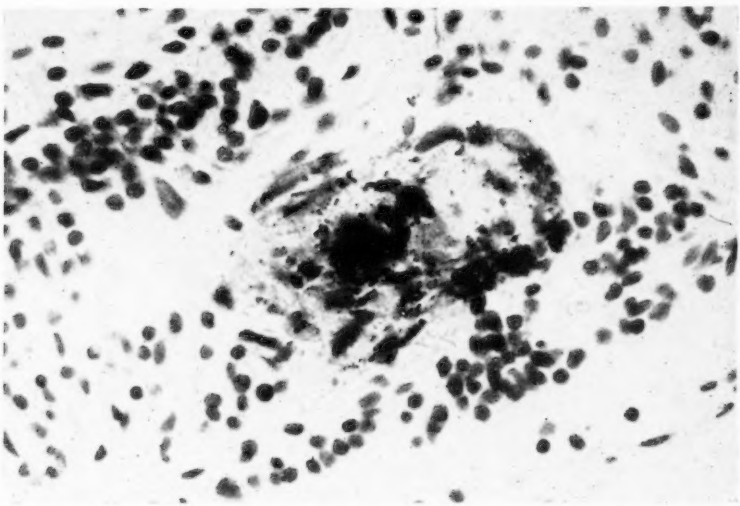

FIG. 19—Case 4. High-power view of a pigment cluster showing hyalo-calcific changes and scattering of free pigment granules in the retina. It is presumed that this represents a degeneration of a pigment cluster.

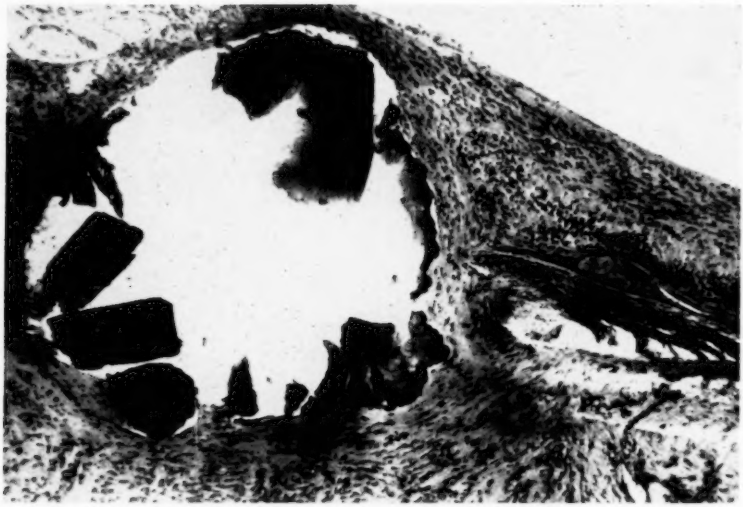

FIG. 20—Case 4. Low-power view of nerve head showing gliosis of retina and massive drusen. Most of the hyaline core fell out during the preparation of the sections.

drusen of the disc which, although a frequent clinical accompaniment of retinitis pigmentosa, has not previously been reported in histologic sections of this condition.

Case 5

Retinitis pigmentosa. Postmortem specimen; paraffin embedding. The patient was a 57-year-old woman who had had night blindness of many years' duration (since early adult life, at least) and had been noted twelve years prior to death to have shown posterior polar cataracts, vitreous opacities, contraction of retinal vessels, some indistinctness of nerve heads, and small areas of pigment in the superficial layers of the retina. The patient died of septicemia, presumably due to a purulent cholangeitis, and the posterior portions of the eye were removed several hours following death.

The histologic specimens showed an absence of the rods and cones and of the outer nuclear layer except for a few abortive cones in the far periphery adjacent to the ora serrata and for a single row of cone nuclei in contact with the outer limiting membrane near the disc and several rows of nuclei of the outer nuclear layer in the region of the fovea (figs. 21 and 22). Elsewhere the outer elements were represented by amorphous debris only (fig. 23). While the apparent disintegration of the rods and cones might be interpreted as postmortem autolysis, the disappearance of the outer nuclear layer indicates that the outer elements had disintegrated ante mortem. There was considerable replacement of the outer retinal layers by neuroglia, but the bipolar cell layer and the ganglion cell layer were normal. The optic nerve showed no atrophy. The pigment epithelium was everywhere intact but showed some depigmentation. Pigment clusters similar to those described in the foregoing cases were to be found in the retina. The choroid showed some sclerosis of its arteries but a normal choriocapillaris. The choroid also showed a patchy lymphocytic infiltration consistent with the terminal septicemia.

Cases of Pseudoretinitis Pigmentosa

I

Figures 24 and 25 illustrate a case of pseudoretinitis pigmentosa. The retina and choroid were from the eye of a young woman who had been blind in this eye since infancy. The other eye was normal. Clinical examination of the eye prior to enucleation showed an opaque heavily vascularized cornea having a central bulge suggestive of staphyloma solidum. The eye was removed on account of pain. These photographs resemble closely those of retinitis pigmentosa in showing disappearance

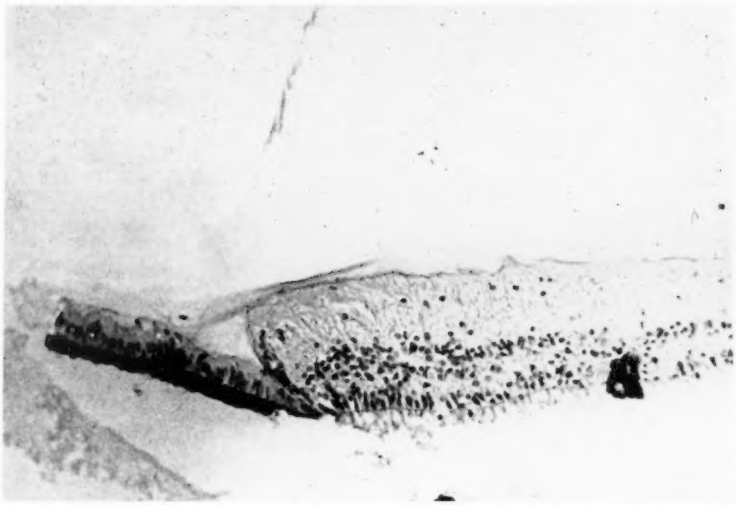

FIG. 21—Case 5. Low-power view of the retina and pars plana of the ciliary body showing pigment cluster extending through external limiting membrane and preservation of abortive cones adjacent to the ora serrata.

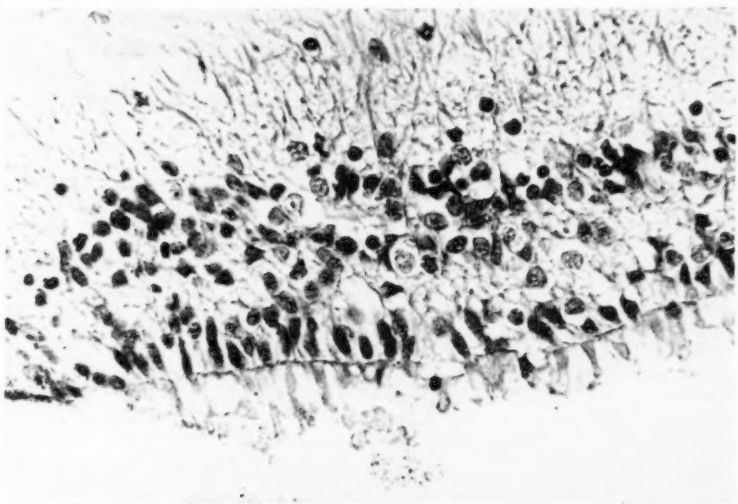

FIG. 22—Case 5. High-power view of same field showing the cones and a portion of the outer nuclear and bipolar cell layer.

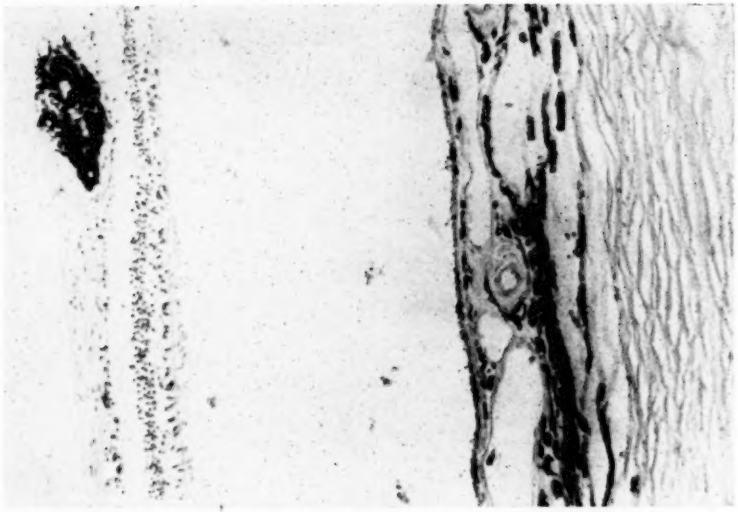

FIG. 23—Case 5. Low-power view of retina and choroid showing a pigment cluster within the retina, almost complete disappearance of the outer nuclear layer, amorphous debris in the region of the rods and cones, absence of any fusion of the retina to the choroid (as indicated by the artefactual separation of the retina), relative depigmentation of the pigment epithelium and moderate sclerosis of the choroidal arteries.

of rods and cones, neuroglia in outer retinal layers, pigment clusters containing hyaline centers (simulating occluded vessels), proliferation of pigment cells into the retina and depigmentation of pigment epithelium. However, examination of the whole eye showed that the changes were less uniform, there was more extensive disorganization of the inner layers of the retina, more extensive alteration in the pigment epithelium, and more fibrosis of the choroid. The presence of genuine chorioretinal fusion, which is thought to be the most reliable criterion in differentiating retinitis pigmentosa from pseudoretinitis pigmentosa, was not a conspicuous feature in this case.

II

Pseudoretinitis pigmentosa. Examination was made of the retina and choroid from the eye of a 59-year-old woman who had had a blind eye with dislocated lens for many years.

The photographs show proliferation of pigment epithelium with formation of pigment clusters having hyaline centers and undergoing hyalo-calcific degeneration similar to that seen with retinitis pigmentosa, but unlike this condition there is marked disorganization of the inner layers of the retina, focal degeneration of the pigment epithelium, genuine fusion of the retina with the choroid, and extensive fibrosis of the latter (figs. 26, 27 and 28).

III

Pseudoretinitis pigmentosa. Figures 29 and 30 show the retina as seen on the flat (unstained) and in cross-section from the eye of a woman with the presumptive diagnosis of old chorioretinitis. The eye was removed because of melanosarcoma of the limbus. No information was available as to the condition of the other eye nor as to whether or not the patient had syphilis.

The appearance of the retina seen on the flat suggests that the pigment follows the course of the vessels, but as indicated in the discussion of retinitis pigmentosa, this is not necessarily the case. The photograph of the cross-section of the retina and choroid shows, in contrast to the findings with uncomplicated retinitis pigmentosa, focal absence of the rods and cones and outer nuclear layer with focal replacement by neuroglia, and genuine fusion of the retina to the choroid with disappearance of the pigment epithelium.

IV

Pseudoretinitis pigmentosa. Two fields of the chorioretinal junction in an eye of a 30-year-old man, who had had a needling of his lenses in childhood and had been blind in this eye since, are shown in figures 31 and 32. The eye was removed with a clinical diagnosis of leukoma and glaucoma. The vi-

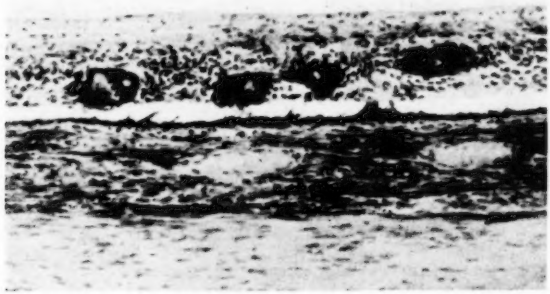

FIGURE 24.

FIGURE 25.

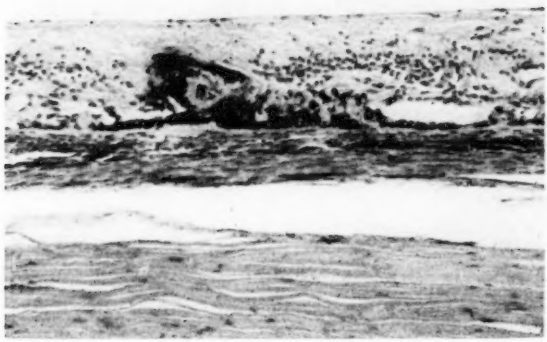

FIGURE 26.

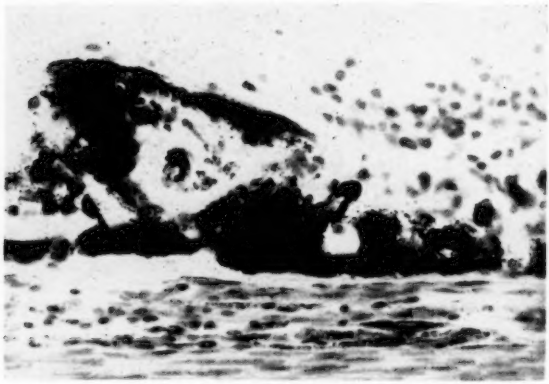

FIGURE 27.

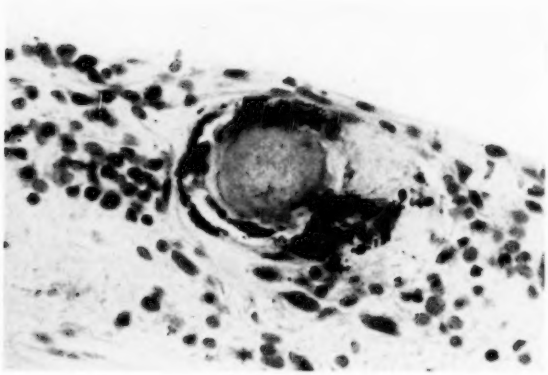

FIGURE 28.

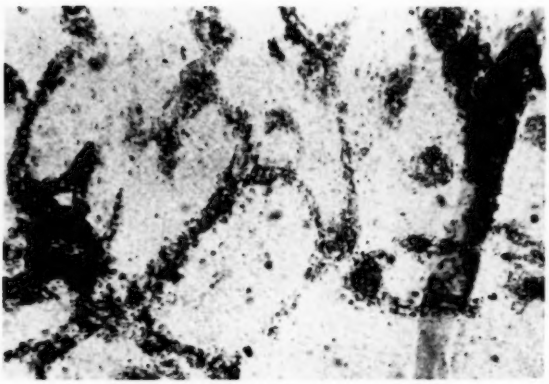

FIGURE 29.

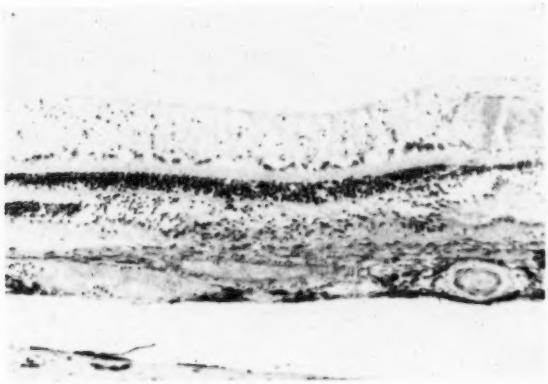

FIGURE 30.

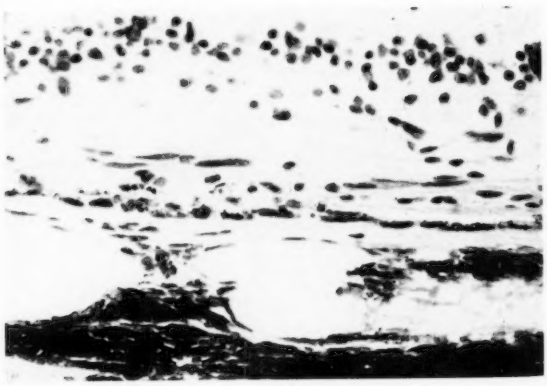

FIGURE 31.

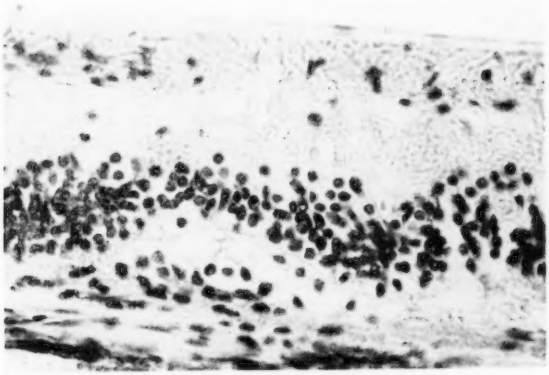

FIGURE 32.

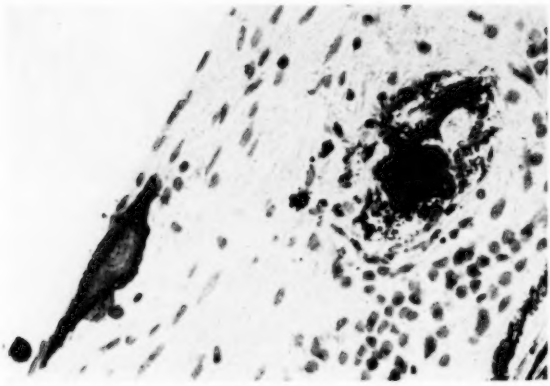

FIGURE 33.

sion in the other eye is said to have been misty, but there was no evidence of retinitis pigmentosa. The field in the first picture is compatible with retinitis pigmentosa, that is, replacement of the outer layers by neuroglia; the pigment epithelial layer is intact (despite some migration of pigment into the retina); the choroid including the chorio-capillaris is well preserved; and there is no real fusion of the choroid to the retina. The field represented by the second photograph, however, is unlike retinitis pigmentosa in that the pigment epithelium has entirely disappeared, and where the external limiting membrane is missing there is genuine fusion of the retina and choroid.

V

Pseudoretinitis pigmentosa. Figure 33 shows a portion of the retina from the eye of a 49-year-old man who had had a posterior dislocation of the lens with secondary iritis and glaucoma in this eye for many years. There was no clinical evidence of retinitis pigmentosa in the other eye. Cross sections of the eye showed loss of the outer retinal layers with extensive fusion of the choroid to the retina, many pigment clusters in the retina, and disappearance of much of the pigment epithelium and the choriocapillaris. The present photograph shows one calcific plaque in the inner portion of the retina and a pigment cluster undergoing a hyalo-calcific degeneration in the midzone of the retina with local scattering of pigment granules. It seems entirely possible that the calcific plaque in the inner retina represents a terminal stage of the changes which are at present occurring in the pigment cluster. Such changes are postulated to occur in both retinitis pigmentosa and pseudoretinitis pigmentosa.

VI

Pseudoretinitis pigmentosa. Retina and choroid from the eye of a 25-year-old man with unilateral congenital cataract, uveitis, and glaucoma are shown in figure 34. The patient's other eye was normal. The photograph shows, in contrast to those of retinitis pigmentosa, profound disorganization of retina, extensive disruption of the pigment epithelium, and widespread fusion of the retina and choroid.

VII

Pseudoretinitis pigmentosa. Retina and choroid of an eye of a middle-aged woman with the presumptive diagnosis of old chorioretinitis are shown in figure 35. The eye was removed with the orbital contents on account of a frontal sinus carcinoma. No information was available to indicate the presence or absence of syphilis. The photograph illustrates the fusion of retina to the choroid with focal absence of the pigment epithelium and extension of what appears to be neuroglia from the retina directly into the choroid.

VIII

"Focal" pseudoretinitis pigmentosa. Figure 36 shows a portion of the retina from the eye of an 18-year-old boy. The eye was removed with a clinical diagnosis of staphyloma of the cornea thought to have been congenital. No further clinical data were available. Histologic examination of the eye showed localized absence of the rods and cones and of the outer nuclear layer over several areas 0.5 to 1.5 mm. in length. Some pigment was to be found

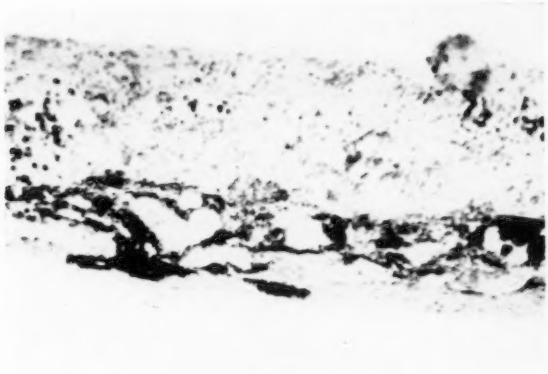

FIGURE 34.

FIGURE 35.

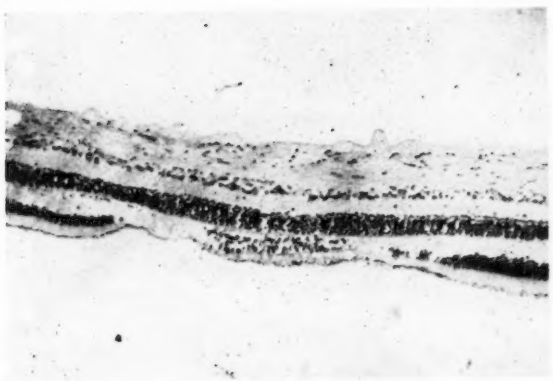

FIGURE 36.

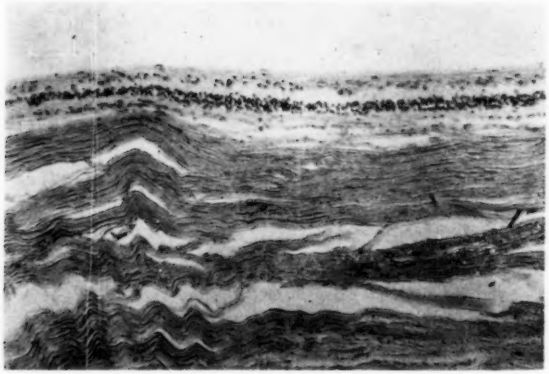

FIGURE 37.

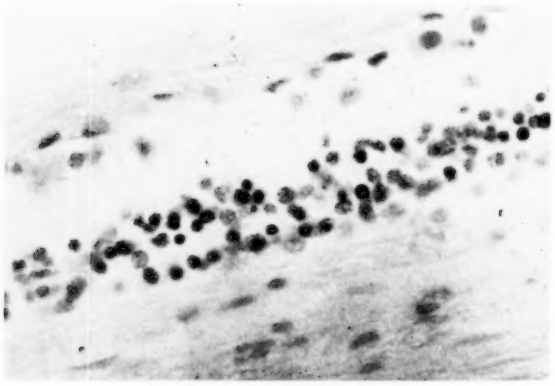

FIGURE 38.

in the retina in these regions, but elsewhere the retina appeared normal. The pigment epithelium overlying the areas of affected retina was relatively depigmented, but the choriocapillaris and rest of the choroid were intact.

Choroideremia

Region from posterior portion of globe from a 76-year-old man who had had symptoms of night blindness since the age of 20 years is illustrated in figures 37 and 38. The clinical aspects of this case have previously been described by McCulloch and McCulloch.[27] The histologic findings, as illustrated in the photographs, consisted of an absence of the rods and cones and outer nuclear layer with preservation of the bipolar and ganglion cell layer, absence of much of the pigment epithelial layer, and disappearance of the choroid over approximately one-third of the posterior portion of the globe. Where the choroid was present, the arteries showed marked sclerosis.

Chorioretinal Changes Following Optico-Ciliary Neurectomy

Section from the posterior portion of the globe of a patient who had had an optico-ciliary neurectomy for pain is shown in figure 39. The histologic findings were variable, and in places the retina and choroid appeared approximately normal, but elsewhere, as indicated in the photograph, there was profound atrophy and disorganization of the retina with thinning and fibrosis of the choroid.

Senile Macular Degeneration

Portion of the retina adjacent to the macula in the eye of an elderly man is shown in fig-

FIGURE 39.

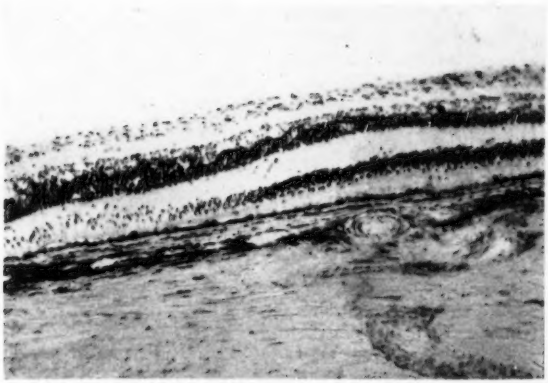

FIGURE 40.

FIGURE 41.

ure 40. The eye was removed on account of advanced glaucoma. The photograph shows the gradual disappearance of the rods and cones and of the outer nuclear layer toward the macula, that is, toward the left. The disorganization of the retina, the proliferation of the pigment epithelium, and the fusion of the retina to the choroid that are frequently found in senile macular degeneration were not evident in this case.

Figure 41 shows the edge of the fovea in another patient who had senile macular degeneration producing an ophthalmoscopic picture of a dark spot in the fovea. The eye was removed for glaucoma. The photograph shows the absence of the rods and cones, proliferation of neuroglia, and disorganization of the retina.

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TREATMENT AND CONCLUSIONS

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The primary pigmentary retinal aberrations with night blindness have up to the present time resisted all attempts at therapy. Since these conditions, in a few instances, were thought to show choroidal sclerosis and attenuated retinal arterioles, the majority of the therapeutic methods proposed have been directed toward the production of vasodilatation with the thought that the degeneration of the retina and choroid might be delayed or stopped could adequate blood supply be provided. The fallacy of this therapy is revealed by Cogan's statement that pathologically retinitis pigmentosa is not characterized by any disease of the retinal arterioles or choriocapillaris. Other forms of treatment are aimed at increasing general body tone or supplying for a supposed lack in vitamins, hormones or growth-stimulating factors.

The literature on the therapy of these conditions simply confuses the reader as to the merits of treatment since the encouraging results reported by one author cannot be duplicated by another. The appearance of new drugs or biologic products down through the years has heralded the appearance in the literature of new treatments for these degenerative diseases. Often the treatment is advocated without laboratory or clinical evidence that such therapy might be applicable. Subsequently, equally competent observers record a contrary opinion with the same therapy.

A careful analysis of the results of treatment by any method shows that there are instances of subjective or objective improvement. However, where the patients treated were followed for any considerable period of time, it was found that the results from treatment were lost. In many instances it apparently was not recognized by the authors that repeated examinations of these same patients without treatment or by treatment with sugar tablets would likewise reveal at times rather marked fluctuations in the clinical findings. Such apparently normal fluctuations in findings are felt by your committee to more than account, in most instances, for the temporary improvement recorded. These fluctuations in vision and fields of vision also make it difficult for an adequate control series to be run parallel with any of the methods of treatment used to lend scientific support to successful results.

No attempt is being made to list all the methods of treatment which have previously been advocated for these conditions, but the following outline includes a representation of the wide variety of substances from which ophthalmologists felt they derived subjective or objective improvement in the ocular status of those afflicted to a degree justifying that the results of treatment be published in the literature.

Presented at the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1949, Chicago, Ill.

PARTIAL LIST OF TREATMENTS ADVOCATED TO DATE IN THE THERAPY OF RETINITIS PIGMENTOSA AND ALLIED CONDITIONS

A. Medical Vasodilators
   1. Inhalation of amyl nitrite
   2. Nitroglycerine by mouth
   3. Miotics as eye drops to produce hypotony
   4. Pilocarpine by mouth
   5. Retrobulbar atropine sulfate (1:1000)
   6. Acetylcholine
   7. Sodium nitrite by mouth

B. Surgical Vasodilatation
   1. Elliot's trephine
   2. Cyclodialysis
   3. Superior cervical sympathectomy

C. Physical Agents
   1. Constant current: galvanism
   2. Roentgen therapy

D. Drugs to Increase General Body Tone
   1. Strychnine subconjunctivally or by mouth
   2. Salt solution subconjunctivally
   3. Calcium phosphate by mouth
   4. Mercury by mouth or inunction
   5. Potassium iodide by mouth
   6. Iron

E. The Hormones
   1. Thyroid gland extract by injection
   2. Female hormone: menformon
   3. Glandulobin: a follicular extract
   4. Testicular extract
   5. Liver extract
   6. B-hormone or melanophore: dispersing hormone of the pituitary

F. Vitamin Therapy
   1. Carotin in oil intramuscularly
   2. Vitamin D: viosterol and calcium phosphate

G. Tissue Therapy: Biogenic Stimulators
   1. Specially prepared cod liver oil by injection
   2. Biogenic stimulators found supposedly in most tissues. After special preparation the tissues are implanted or its extracts injected under the conjunctiva or skin. Tissues used are:
      a. Skin, liver, spleen, subcutaneous tissue, muscle, cartilage, placenta, retina, sclera, choroid, tarsus, etc.
      b. Vegetable products such as leaves of the aloe and pea sprouts

To this list will undoubtedly be added additional unproved forms of treatment, but it is hoped that sooner or later the day will arrive when a ray of hope may be given these unfortunate people by the discovery of the inherited factor which results in the specific metabolic disorder producing this group of diseases.

The medical profession has long ago accepted the fact that retinitis pigmentosa and its allied conditions are degenerative as well as inherited diseases and, as such, are not amenable to treatment. Let us recall, however, that with advances in medical knowledge other diseases formerly similarly catalogued as degenerative and hereditary in nature and not amenable to treatment have been removed from the incurable list and can now in many instances be controlled, if not cured. For example, the Schüller-Christian-Hand syndrome is now effectively treated by roentgen therapy to the involved areas when seen early. The same might likewise be said about diabetes, gout, pernicious anemia, familial hemolytic jaundice, etc. It is granted that as yet we are unable to influence the individual genes to prevent the transmission of undesirable inherited characteristics once the pattern is set, but if these inherited conditions are recognized early before irreparable damage has been done to the individual and known corrective measures initiated to compensate for the metabolic disorder, then a useful individual results. If treatment is started late when the degen-

erative changes have altered function, it is then of little value.

We feel justified, therefore, in criticizing the methods of selecting cases for treatment as presented in the literature to date. Patients have been chosen who were already far advanced in their degenerative process, when nothing but poor results should be expected. A more accurate evaluation of treatment would be obtained by investigating the families of afflicted patients and treating especially those members showing the earliest recognizable characteristics of the disease. If the treatment has merit, further degeneration should be prevented.

Another method of attack is that from the eugenic point of view. This prophylactic form of therapy has as yet a very limited practical application in humans but is accepted practice in domesticated animals. It may never have much influence on our problem because of its unpopular implications, but at the same time the information which the heredity clinics (or geneticists) are able to give from a study of the family should be available to those desiring to use it. To give such information requires that each family be studied carefully to permit an expression of the exact mode of inheritance.

The one method of treatment usable today in selected instances of the primary pigmentary retinal aberrations is that of cataract extraction. It, however, has been passed down through the literature as an accepted fact that in retinitis pigmentosa, operations either for the removal of cataract or for glaucoma have a great tendency to form fibrous tissue and thus do poorly. An attempt has been made by your committee to re-evaluate this statement in the light of modern medical experience. A questionnaire was sent out to many ophthalmologic centers and to a sampling of the ophthalmic surgeons of the country in an attempt to obtain this information. It was found that relatively few patients having retinitis pigmentosa have been operated upon by any one individual or institution. However, from the information received it is possible to state that cataract extraction associated with retinitis pigmentosa offers no complications different from those occurring in the normal eye operated upon for cataract.

Further, the questionnaire reveals that the visual results of cataract extraction in retinitis pigmentosa may be very disappointing to the surgeon and at times to the patient. This is due to a number of factors.

1. The surgeon is likely to compare the resultant vision with that obtained in cataract extraction in otherwise normal eyes. On the other hand, as aptly stated by Courtney, "These retinitis pigmentosa patients had already adjusted themselves to poor vision and had developed a facility for using what vision they had, so that following the cataract extraction with visual improvement they are able to live quite useful lives."

2. It is usually only late in the course of the disease that lens changes are so advanced that cataract extraction seems to be indicated. At this stage the field of vision is small, and even though the central acuity of vision may be improved, the operation may not greatly increase the patient's visual efficiency, due to the restricted field.

3. Unless the patient's hopes for visual improvement are restrained preoperatively, he is likely to expect too much from the operation.

On the other hand, of the 158 cases operated upon for cataract in the presence of retinitis pigmentosa which your committee was able to collect through the questionnaire, the operating surgeon interpreted the results of surgery as giving definite and in many cases startling visual improvement in 139 instances and no improvement in 19. Of the forty-two surgeons who ex-

pressed an opinion as to the advisability of doing cataract extraction in selected cases of retinitis pigmentosa, only two felt that cataract extraction was useless. Needless to say these data also point out that the operation has no retarding effect on the progression of the degenerative process.

PROGNOSIS

The prognosis for retention of vision is not too difficult to foretell in the group of patients with congenital stationary night blindness, for in this group the patient can expect to retain his present vision throughout life if other pathologic change can be eliminated. This group does, however, show instances of related defects such as amblyopia, nystagmus, myopia and neurologic defects which cannot always be foreseen without a careful genetic study and follow-up of each patient.

The group of diseases classified under delayed or progressive primary chorioretinal aberrations are much more difficult to prognose as to visual retention but certainly the majority will show a rather steady progressive visual impairment and field loss. Just how rapidly these changes may be manifest is best predicted by a careful examination of not only the affected individuals in the family but the apparently unaffected ones as well. Only through these means of determining the nature of the pedigree in individual families can reliable advice be given as to the nature of the progress and mode of transmission of the degenerative disease to the next generation. Although there is some variation in the progression of the degeneration within any one family, there is greater similarity between related affected individuals than exists in comparison to unrelated affected individuals. The later in life an individual first notices his visual handicap, the more likely is he to have useful vision in later life.

The prognosis with cataract surgery in retinitis pigmentosa when the vision is as poor as 20/200 is surprisingly good. The operation offers no more complications than does cataract extraction in ordinary individuals. There apparently is no more tendency for glaucoma or retinal detachment to follow the surgery than is usually experienced. Cataract surgery, however, has no retarding effect on the degenerative disease.

DISCUSSION

SIR STEWART DUKE-ELDER
LONDON, ENGLAND
BY INVITATION*

As Dr. Leinfelder has said, the classification of this group of diseases is difficult because of the lack of pathologic knowledge. This makes it necessary to base the classification on clinical signs and symptoms — always a dangerous procedure but in this case, of course, quite necessary. The leaders of this symposium I feel must definitely be congratulated on their courage. I think they have succeeded remarkably well. The essential clinical symptom they have chosen is night blindness. This is tantamount to grouping together those aberrations of the fundus which affect the rods rather than the cones or, topically speaking, grouping together those aberrations which are located primarily or mainly in the periphery rather than in the central region. I am just a little uncertain whether this is a sufficiently fundamental symptom whereupon to base a classification which includes primary choroidal aberrations.

For example, in a classification of this nature there might be a case for including primary choroidal sclerosis among the progressive choroidal atrophies. It is a primary atrophy of the choroid and it is progressive. As you know, it is a disease of quite unknown etiology, characterized by a replacement of the muscle tissue in the choroidal vessels by fibrous tissue, with subsequent degeneration of the intervascular stroma followed by secondary degeneration of the outer retinal layers and pigmented epithelium so that the white fibrotic atrophied choroid is clearly seen; it is associated frequently with the bone-corpuscular type of pigmentary deposition characteristic of retinitis pigmentosa. It is not associated with general vascular or retinal disease.

Although this condition does not usually appear until after midlife, it may occur in the young. It is frequently familial, and into the same heredity there sometimes comes retinitis pigmentosa. If the disease is diffuse — and I think this is the important point — an early and constant symptom is night blindness which may progress to total blindness; but if, as may be the case, it is limited to the central area of the retina, night blindness does not, of course, occur. There might be a case for considering this disease within this classification. I am by no means insisting that it should be, although it is primary atrophy of the choroid; but if we did, it would be difficult in any classification to include the diffuse form which is associated with night blindness and to exclude the central form which is not associated with night blindness.

It might be argued, therefore, that when there is a primary degeneration of the retinal rods, the group of aberrations so designated will be homogenous and allow, if you will, of legitimate classification. So far as this goes, groups I

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and II-A and B in the ones presented are excellent, but it may be asked whether group II-C is legitimate in our classification, that is, choroidal atrophies, for here night blindness, which forms the basis of our system, may perhaps be a secondary topographical incident and may not be fundamental.

The trouble is, of course, that in our present state of ignorance no one can be sure whether this concept is right or wrong, but there is one thing quite definite—that the presentations of the group as put forward this morning should go far toward clarifying our knowledge of a very difficult and complex subject.

f f f

QUESTION AND ANSWER PERIOD

DR. FRALICK, Moderator: I am not going to attempt to summarize the presentations of the previous discussers. I simply wish to refer you to the scientific exhibit, where Dr. Falls has prepared a very excellent summary. The committee wishes, however, to urge each member of the Academy to be on the constant lookout for instances where pathologic material referable to these conditions may be obtained. The paucity of pathologic material is in part responsible for our limited knowledge about most of these conditions, for we secure eyes only late in the course of the disease.

We will now have the questions and answers.

Dr. Leinfelder, should not retinitis pigmentosa sine pigmento be included in the outline?

DR. LEINFELDER: As you heard in Dr. Carroll's delineation of the signs and symptoms of the disease, there is a great variation in the pigmentary pattern of various cases of retinitis pigmentosa, in any one hereditary group or in various hereditary groups. We may find under some circumstances siblings who have a great amount of pigmentation in the fundus relatively early in the disease, while in other patients there is very little pigment even later in the disease. In discussing this condition in the committee meetings it was decided that the best evidence seems to indicate that retinitis pigmentosa sine pigmento is not a definite entity but rather an accidental one which might exist throughout the course of the disease or which might represent only an early stage of the disease. It was, therefore, not made an individual group in the classification.

DR. FRALICK: Thank you, Dr. Leinfelder. Dr. Carroll, the question is asked, "If the nerve head in choroideremia is normal in appearance, why does it not stand out sharply in contrast to the white fundus?"

DR. CARROLL: The nerve head in choroideremia sometimes does stand out fairly prominently as compared to the very white background. It is rather pink as compared to the very white background. Some of my drawings may not have shown the pink nerve, but that was the artist's fault.

DR. FRALICK: What practical office procedure for measuring dark adaptation is advised?

DR. CARROLL: The simplest one is to take the patient into a room where there is diminished illumination. Nettleship used to do that, and it is still a very good procedure. He would test the fields on himself first (he apparently had normal fields) and then he would do it on the patient. You can do it roughly just by confrontation in a room with marked diminution of illumination.

DR. FRALICK: Do these studies throw any light on the disturbance of dark adaptation following exposure to ultraviolet irradiation, such as fluorescent lamps?

DR. CARROLL: Not that I know of.

DR. FRALICK: Why do you not use the term tapetoretinal in your discussion?

DR. CARROLL: Because the human eye has no tapetum. The tapetum is very important to dogs and cats, but we have none. It is a long name which I think we can throw out.

DR. FRALICK: How can you distinguish between juvenile amaurotic idiocy and retinitis pigmentosa?

DR. CARROLL: That may be quite difficult. However, there are certain general differences. In juvenile amaurotic idiocy the changes are more likely to be in the macular region. The contraction of the retinal vessels is much less marked and the peripheral pigment changes are much less likely to be marked. The night blindness in retinitis pigmentosa is much more prominent. The dementia that occurs, at least eventually, in juvenile amaurotic idiocy is much greater, and the mental deficiency progresses much more than it does in retinitis pigmentosa.

DR. FRALICK: Will you clarify the term "atypical retinitis pigmentosa"?

DR. CARROLL: First we have to say what typical retinitis pigmentosa looks like, and I think we all agree on that. In the atypical

form there is one type we see in the Laurence-Moon-Biedl syndrome, in which the macula is often involved early, but these patients do have night blindness. Then you have retinitis pigmentosa sine pigmento, which I think many people used to call tapetoretinal degeneration. Then we have all grades of depigmentation, so that you can see the choroidal vessel markings very prominently.

DR. FRALICK: Please give the differential diagnosis between hereditary choroidal sclerosis and choroideremia.

DR. CARROLL: In choroideremia we see what appears to be marked choroidal sclerosis. The affected individuals do have night blindness, which is usually the first symptom. Most of these patients have had night blindness as long as they can remember. In hereditary choroidal sclerosis, as Sir Stewart has just said, they are more likely to have loss of central vision first. The night blindness is a minimal complaint, comes on fairly late and occurs, I believe, only when there is generalized choroidal sclerosis.

DR. FRALICK: Thank you, Dr. Carroll. Dr. Falls, what form of heredity is shown in retinitis pigmentosa when three brothers and a sister with unrelated parents have the disease. Parents and grandparents are apparently free of the disease.

DR. FALLS: It is impossible to state with the evidence available. Three possibilities may be considered.

1. We are dealing with the simple autosomal recessive form of the disease. I remarked previously that the simple recessive form of inheritance is most difficult to prove or diagnose.Consanguinity is merely an important aid to establishing this mode of inheritance. It is also most imperative that the family pedigree be known in all its most minute detail and as far back as possible. This is difficult since most of us do not know the maiden names of our grandmothers. This is particularly true of most of our urban population. We are no longer family conscious! Many of us, on the other hand, carry genes for rare recessive diseases, such as retinitis pigmentosa, and mere chance mating of two such unrelated heterozygous individuals can have offspring possessing the homozygous state and thus be affected. In the family mentioned, the fact that four children are affected is not incompatible with recessive inheritance, since each child has one chance in four of being affected. The mere vagaries of the laws of chance would permit many such unusual combinations.

2. Mutation may have occurred in the germ plasm of one of the two parents. If the mutation occurred in the primordial germ cells and was a dominant mutation, then many of the offspring could be affected. If, however, the mutation occurred late in the formation of the parental germ plasm, then affected offspring would appear with extreme rarity or only once. Dr. Algernon Reese, in his recent article on "Hereditary Retinoblastoma," mentioned that lightning (mutation) seldom strikes twice. It must be agreed that by far the greater majority of families having a sporadic case of retinoblastoma will not have further affected offspring, but there is the occasional family in which the disease will occur with frightening frequency. Consequently, when giving genetic advice to such parents we cannot be dogmatic since we have no way of knowing where the mutation has occurred.

3. The last possibility is that the disease may be the result of an environmental noxious agent exerting its influence during pregnancy. Retinal pigmentary disturbance has been recorded as a sequela of maternal rubella. It is important, therefore, that we inquire carefully into the maternal health during pregnancy.

DR. FRALICK: Dr. Falls, what single book would be best for the study of heredity and transmission of ocular diseases? Publishers?

DR. FALLS: Dr. P. J. Waardenburg has an excellent monograph on the hereditary ocular diseases, "Das Menschliche Auge und Seine Erbanlangen," Bibliographia Genetica, vol. 8, Gravenhage Martinus Nijhoft, 1932.

Dr. Arnold Sorsby's "Modern Trends in Ophthalmology," vols. 1 and 2, Paul B. Hoeber, New York, has excellent chapters on hereditary ocular diseases. Dr. P. J. Waardenburg has contributed to both volumes.

Sir Stewart Duke-Elder's "Textbook of Ophthalmology," vol. 1, C. V. Mosby, St. Louis, has a very readable chapter on heredity.

DR. FRALICK: Should the following couple have children? Each has retinitis pigmentosa with macular degeneration and vision of finger movements or hand movements. Each of these individuals is in the early twenties. The families are not related. No similar condition exists in either family tree as far as can be traced. What are the chances that their children will have the same disease?

DR. FALLS: The only answer a physician can give these individuals is that they are not to have children. When two individuals affected with the recessive type of retinitis

pigmentosa marry, then all of their children will be affected.

From a cold and purely scientific viewpoint it would be interesting to see what would result from the union. If the parents have the same gene, then all the children would be affected; but if the genes are unlike, then none of the offspring will be affected. The latter would, however, be carriers (heterozygous) for either of the genes of their parents.

DR. FRALICK: There are a number of other questions which I shall attempt to answer.

Question: Concerning Filatov's tissue therapy, what efforts have been made to confirm his results? What is the result of such efforts?

Answer: Dr. Dan Gordon has made an extensive study of Filatov's tissue therapy and his results are published in the 1948 TRANSACTIONS of this society. My interpretation of his results in the treatment of retinitis pigmentosa was that they were uniformly unsatisfactory.

Question: In my experience, sufficient vitamin D and sufficient calcium have benefited patients suffering from retinitis pigmentosa and myopia. There was definite subjective and objective improvement, including improved night sight, for years. Could you please tell us if you have had any experience with the treatment?

Answer: I have had no personal experience with this form of treatment. From the literature we gather that there is no uniformity of opinion among equally competent observers that this or any other form of treatment is of any value in retinitis pigmentosa. We have to remember that there is considerable fluctuation in findings, both in vision and in visual fields in untreated patients, and it might be that these beneficial effects obtained by vitamin D and calcium are on that basis. Only by having an adequate control series and by following these patients over a considerable period of time can we determine whether it is simply a remission or fluctuation and not the result of treatment.

Question: I should like to have information on the Filatov tissue treatment. How is tissue obtained? How is it sterilized? How much is used at a time? How and where is it implanted? In what intervals and how many times is it repeated?

Answer: This question involves a great deal of detail that would take longer to discuss than our time allows. I would refer you to the original articles of Filatov, which may be read in English. (Am. Rev. of Soviet Med., 2:53-66, 1944).

Question: I have been impressed by the needless cruelty of many men in giving the patient a very poor prognosis in retinitis pigmentosa. Most patients are told that they will go blind and even deaf as well. In my series of several hundred cases very few have no light perception and most have some degree of Snellen vision. Is this your opinion?

Answer: It is simply necessary to inform the patient that the progress of the disease is usually slow and that few people actually become blind. The earlier in life the night blindness appears, the more likely is serious visual impairment to occur during the active years of the individual. I do not believe there are available statistics showing the degree of visual loss in different age groups or showing what percentage of people do actually become blind during their lifetime.

Question: What type of cataract extraction seems to be indicated in retinitis pigmentosa?

Answer: The questionnaire sent out by your committee revealed that the intracapsular extraction is the preferable one. This, of course, may be a reflection of the trends of the present methods of cataract extraction in general and not in regards to cataracts in retinitis pigmentosa specifically.

Question: What would you consider the indications for cataract extraction in retinitis pigmentosa?

Answer: This was also answered in the questionnaire in many instances. The concensus of opinion was that if the patient's vision had decreased to 20/200 or 6/60, cataract extraction was indicated. Even where the paprovement could be contemplated.

NUCLEAR DEAFNESS AND THE NERVE DEAF CHILD: THE IMPORTANCE OF THE RH FACTOR

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THE etiology and pathologic anatomy of nerve deafness have not received the intensive study which has been accorded them in conduction deafness. Recognition of maternal rubella as an etiologic factor in perceptive deafness was the first major contribution to the problem in more than 40 years.

The present study deals with another new potential etiologic factor. Varying degrees of hearing impairment and deafness have been noted in certain children as presumptive sequelae of kernicterus. The term "kernicterus" has been used to describe the severe central neuropathologic complications of erythroblastosis fetalis or hemolytic disease of the newborn. It is now recognized that Rh factor incompatibility is the primary cause of erythroblastosis fetalis. Thus, the recently isolated phenomenon of iso-immunization (due to the Rh factor) may yield a hitherto undescribed etiologic factor of a central variety of nerve deafness.

The pathogenesis of kernicterus and audiometric analysis of this group of cases point to a central rather than a peripheral localization for the perceptive deafness. This central area is predominantly in certain of the nuclear structures of the brain, hence, the term "nuclear deafness."

This presentation is a preliminary analysis of case material which has been selected because of the significant correlation of perceptive deafness, erythroblastotic history and clinical findings suggestive of kernicterus. No attempt at a true statistical analysis has been made in this study. The present group of cases is a somewhat random sampling of a larger group which will be subjected to statistical analysis in the near future. Hence, the present study is not to be measured on statistical grounds but is primarily presented to stimulate further investigations into the potential auditory sequelae of erythroblastotic kernicterus.

ETIOLOGIC ASPECTS OF INFANTILE NERVE DEAFNESS

Our present knowledge of the etiology of infantile nerve deafness is extremely scant and confused. The discussion of such etiology in almost every modern textbook of otolaryngology is almost identical with the list of causes described by Politzer[12] over fifty years ago. Archaic classifications have been handed down from textbook to textbook so that extremely rare and dubious causes are magnified by tradition into apparently important factors in the etiology of infantile nerve deafness.

In the United States Census Bureau Report of 1920, an analysis of the causes of deafness in 35,000 individuals listed 53.5 per cent as unclassifiable. More accurate otologic analyses of the populations of schools for the deaf have given

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percentages of 20 to 30 as unclassifiable or unknown. Differential diagnostic methods in this confusing field are admittedly inaccurate.

Typical of the backwardness of otologic thought in this one subject is the fact that the term "deafmutism" or "Taubstummeheit" still persists in the literature. "Deafmutism" is not a medical or otologic term. It is simply a delineation of a certain group of profoundly deafened children who because of their deafness have been unable to acquire adequate speech. It is, therefore, unscientific to use such a pedagogic term in medical diagnosis.

The recent discovery that rubella (German measles) in the mother during the first trimester of pregnancy might cause deafness in the child was the first major contribution to the etiologic analysis of infantile nerve deafness in the present century. This contribution is truly monumental. The statistical significance of maternal rubella in congenital perceptive deafness is far greater than most of us realize. This subject is being studied at the present time and will be reported in the near future. In a preliminary analysis of 900 questionnaires dealing with profoundly deafened children, in 166, or 18 per cent, a history of maternal rubella in the first trimester of pregnancy was obtained.

It is not within the province of the present study to deal at length with the vast problem of etiologic factors in congenital deafness. It is of interest, therefore, to relate the deafness due to the neuropathologic sequelae (kernicterus) of Rh incompatibility to the vast "unclassifiable" field of infantile nerve deafness.

DEVELOPMENT OF THE PRESENT STUDY

The present study was prompted by two apparently separate and unrelated groups of observations.

1. Several years ago, a study of the otologic status of cerebral palsied children was undertaken at the Children's Hospital in Los Angeles. One reason for the study was the fact that many of these children had speech defects for which a cause was sought. Another reason was the fact that it had been observed by some clinicians that the athetoid variety of cerebral palsy was frequently accompanied by deafness and hypacusis of varying degrees. Phelps,[10,11] in 1941, noted a definite incidence of defective hearing in the athetoid group. He concluded that this hearing loss was of a central nature and did not represent total deafness. He observed that it was limited to certain pitch ranges so that, while the child was able to hear some sounds, he was not able to hear others. The result of this was a type of defective speech which was characteristic of the athetoid child.

In our study of the cerebral palsied group, we found a similar incidence of deafness and hypacusis in the athetoid subgroup. It was of even greater interest, however, to note that this same subgroup revealed a large percentage of patients with histories suggestive of erythroblastosis fetalis or hemolytic disease of the newborn. It was then found that most of these children were the Rh positive children of Rh negative mothers. Further serologic studies revealed that most of these mothers continued to show anti-Rh antibodies in their blood stream many years after the birth of the child. These findings will be detailed in the case reports.

2. The second reason for the present study was based upon random observations in the routine examination of a large group of perceptive deafened children. It was noticed that there was a high incidence of histories suggestive of erythroblastosis in these perceptive deafened children without symptoms of cerebral palsy. In the study of a questionnaire sent to the mothers of 900 profoundly deafened children, histories

highly suggestive of erythroblastosis fetalis with jaundice at birth were obtained for a group of patients much larger than that which could be expected in the normal population. Some of these numerical aspects will be presented later.

ERYTHROBLASTOSIS AND RH INCOMPATIBILITY

Nine years ago (1940), Landsteiner and Wiener[8] described a new agglutinogen in human red cells which they termed the Rh factor because a similar agglutinogen was found in the red cells of the rhesus monkey. This observation began to explain many hitherto unexplained problems apparently related to iso-immunization. It also began to cast a light on the mysterious disease which had been called erythroblastosis fetalis. The latter serious condition of the newborn had a high mortality rate (30 to 40 per cent roughly) and had been called many names, among which were septic jaundice of the newborn, septic anemia of the newborn, and congenital hemolytic anemia.

A tremendous amount of literature has appeared on the Rh factor. Briefly, all persons whose red cells lack the Rh agglutinogen are spoken of as being Rh negative. All individuals whose cells possess the Rh agglutinogen are considered to be Rh positive. Rh negative individuals do not normally have anti-Rh agglutinins in their plasma. However, Rh negative individuals are liable to form antibodies if Rh agglutinogen is introduced into the circulation. Rh agglutinogens can be introduced into the circulation in one of two ways. They may be introduced in persons of both sexes when transfused with Rh positive blood. Secondly, they may cross the placenta in women pregnant with an Rh positive fetus, the fetus having inherited the Rh agglutinogen from the father. It became obvious, therefore, from the transfusion standpoint that the presence of anti-Rh agglutinins, developed as a result of transfusions or of pregnancy, would render an individual incompatible for a subsequent transfusion of Rh positive blood. Now, since 85 per cent of bloods are Rh positive, the chances of incompatible blood being given in such a case are great unless Rh negative blood is used. It is, therefore, easily seen how important is the detection of anti-Rh antibodies in the circulating blood of an Rh negative woman. Such antibody recognition will distinctly contraindicate transfusion with Rh positive blood and will cast a great light upon any study involving the Rh factor as a cause of defects in the newborn.

Erythroblastosis fetalis or congenital hemolytic disease has been known for years, and the term was developed because these infants universally showed large numbers of erythroblasts or nucleated red cells in the circulation for a longer period than was normally recognized as necessary for the physiologic adjustment of hematopoiesis. Children recognized as having this disease were usually seen in one of three states. The first state was referred to as congenital hydrops fetalis and was characterized by an infant who was born either dead or moribund with generalized edema. The second variety was termed icterus gravis neonatorum and was characterized by a child with complete jaundice at birth or within 24 hours after birth. The third variety was termed congenital anemia of the newborn in which a child was normal at birth and developed severe anemia within a few days.

It is accepted today that congenital hemolytic disease is produced by the presence of Rh antibodies in the maternal blood. If an Rh positive husband is homozygous for Rh, an Rh negative woman can expect all of her children to be Rh positive. If her husband is heterozygous for Rh, an Rh negative woman can expect half of her children to be Rh

positive. Thus, during pregnancy, it is theorized that the blood of an Rh positive fetus escapes from the placenta into the maternal circulation. If the mother is Rh negative, antibodies against these cells may be produced by the mother. Such antibodies may then pass from the maternal blood into the fetal circulation and destroy Rh positive cells of the fetus by combining with them. The end result of this pathologic state is erythroblastosis fetalis.

Unless the mother has been previously transfused with Rh positive blood and has developed antibodies, or has had a previous pregnancy resulting in an abortion, a first born child rarely will have erythroblastosis fetalis. It is rare to find sufficient concentration of antibodies during the course of a single pregnancy to harm that particular fetus. However, once the concentration of antibodies responsible for hemolytic disease in the newborn has been developed in the mother, all subsequently conceived Rh positive fetuses will probably suffer from hemolytic disease as a result of the iso-immunization and the maternal antibodies that have persisted from the previous pregnancy.

The incidence of hemolytic disease is not too well established numerically at the present time. This is undoubtedly due to the fact that recognition of the Rh factor is recent and that statistical studies on this subject will take many years to reach a fine degree of accuracy. However, most authorities today predicate the ratio of hemolytic disease between 1 in 150 and 1 in 250 births. Dr. Edith Potter[13] reports from the Chicago Lying-In Hospital that during the year 1944 the ratio was 1 in 21 babies born to Rh negative women or about 1 in every 150 births. In 1945, Dr. Louis Diamond[3] reported from Boston a similar incidence of 1 in 150 births.

KERNICTERUS: ITS NATURE AND SIGNIFICANCE IN ATHETOSIS

Orth,[9] in 1875, first described kernicterus as a pathologic entity. In 1903, Schmorl[14] coined the term "kernicterus" to designate jaundice of various nuclear cranial masses. The structures most commonly involved were the caudate, lenticular, subthalamic and dentate nuclei, the thalami, the mamillary bodies, the cornua Ammonis, the nuclei of the cranial nerves, the superior and inferior olives and certain portions of the cerebellar cortex, as well as the posterior and anterior horns of the spinal cord.

In 1933 Zimmerman and Yannet[18] made the following conclusions:

> Jaundice of these structures is not a disease *sui generis,* but occurs as one of the lesions that may be found in new-born infants with severe jaundice and only in a comparatively small number of these. . . . The lesion has never been observed in adults; indeed, there are no reports of it beyond the neonatal period.
>
> Jaundice of the nuclear masses of the brain is most frequently, if not exclusively, associated with icterus gravis neonatorum. Obstructive jaundice has yet to yield an instance of kernikterus.

In a paper on the neurologic sequelae of erythroblastosis fetalis, Docter[4] states:

> The term kernicterus is being used to describe not only an anatomic pathologic lesion, but also the clinical manifestations of this lesion as observed in the living patient. If the patient survives the neonatal period there develops immediately, or weeks or months later, evidence of damage to the basal ganglia, that is, extrapyramidal spasticity, athetoid and choreiform movements, and emotional instability plus more or less marked mental retardation.

Klingman and Carlson[7] found the most common symptoms of kernicterus to consist in convulsions, spasticity, apathy or lethargy, restlessness, increased crying or difficulty in feeding in the early stage of the disease. The later symptoms consisted in delayed motor

functions, gradual progressive spasticity, inco-ordination, choreo-athetosis, ataxia, hypotonicity and retardation of mental development.

Older, pre-Rh theories on pathogenesis attributed the disease to (1) a peculiar attraction of bile pigments for ganglion cells, (2) primary damage of ganglion cells by substances such as bile salts with secondary cellular pigmentation, or (3) ganglion cell damage due to ischemia or trauma with subsequent pigmentation of the cells.

However, this older concept is apparently refuted by the new work of Dublin.[5] He states in a paper read in 1948:

> Previously it was thought that kernicterus in infants is found only in erythroblastosis fetalis. More recently, kernicterus has been reported unassociated with erythroblastosis. . . . Of six cases of kernicterus of the newborn observed during the past year, only two showed erythroblasts in appreciable numbers in smears of peripheral blood or in postmortem histologic sections, in pulmonary vessels. Of the remaining four subjects, two presented obstructive congenital anomalies of the bile ducts, one showed intrahepatic obstruction from cryptopathic parenchymal degeneration, and one presented an infectious or toxic hepatitis.
>
> Golden yellow pigment appeared in corpus striatum, thalamus and hippocampus, in inferior olivary, dentate, and vestibular nuclei, and in a patchy form in the cerebral cortex. This pattern of distribution is the same as that of degeneration in cerebral anoxia. . . .
>
> In every case, the yellow pigment was found microscopically, in areas grossly tinted, encrusting degenerated ganglion cells, about vessels, and less distinctly throughout the intervening tissue. . . .
>
> It is evident that: (1) injury of nerve tissue, (2) hyperbilirubinemia, and (3) excessive permeability of the blood-brain barrier for the pigment, will, regardless of the age of the subject and the original causes of the three factors, result in kernicterus. The latter has been reported by Rutledge and Neuberger in jaundice plus multiple foci of degeneration in the brain, apparently arteriosclerotic. . . .
>
> The mechanism of injury of nerve tissue in kernicterus associated with erythroblastosis fetalis but not with difficult labor or other sources of injury, is not clear. Two factors play a part: (1) Brain tissue is injured by anti-Rh, anti-A or anti-B immune bodies. Rh antigen has been demonstrated in body tissues, but, being soluble in alcohol, but not in water, is not found in body fluids. (2) Anemia due to destruction of erythrocytes results in general, including cerebral, anoxia.

Windle and his co-workers[16] have shown that certain marked alterations in brain structures will occur after asphyxiation at birth. They recently stated that experimental asphyxiation followed by successful resuscitation of guinea pigs at birth produced neuropathologic changes of variable degrees of severity; the thalamus, cerebral cortex, tegmentum and spinal cord were often severely damaged.

KERNICTERUS AND DEAFNESS: THEORETIC ASPECTS

Inasmuch as a high degree of correlation was found between athetoid cerebral palsy, kernicterus and a symmetric bilateral perceptive deafness, a presumptive localization of the auditory lesion was made in the central auditory pathway. The lesions described in kernicterus were primarily confined to the chief ganglion and nuclear masses.

In view of the bilaterality of the deafness and its audiometric symmetry (both for frequency and intensity), the theory that the cause lies in a central lesion is more logical than that it lies in a cochlear lesion. It would be highly fortuitous if icteric deposits in two cochleas were so identical in location and degree that bilateral symmetric perceptive impairments would be obtained in most of these cases.

Even though our knowledge of the intracranial auditory pathways is incomplete, certain facts are reasonably solid. The bilateral cortical representation of each cochlea is well accepted. Furthermore, a certain degree of point to point correlation is also well established. Spatial representation of the cochlear geography upon the auditory cortex was re-

cently substantiated by the work of Walzl and Woolsey.[15]

Of further interest in this analysis is the animal work of Culler,[2] who in studying the medial geniculate bodies found that the several frequencies traverse the geniculates by differential pathways. Thus, the topographic distribution of frequencies which begins in the cochlea persists at least as far as the geniculates.

The exact routes of the pathways, however, are still highly controversial. It would not be unlikely for a brain-stem lesion to be so located that icteric deposits, either in cochlear nuclei or superior olives, could produce some degree of bilaterally symmetric perceptive hearing impairment.

In regard to this problem, Dr. Olaf Larsell stated, in a personal communication:

> Investigations are now under way in our laboratory on the superior olivary complex from the cochlear nuclei of both sides, probably accounting in large part for the bilaterality of hearing. The superior olive and the cochlea are both very sensitive to noxious elements in the blood stream. Presumably, both would be easily injured by anoxia due to retarded onset of respiration at birth, exacerbated by the erythroblastosis fetalis or the latter condition alone conceivably could be responsible; if the deafness is unilateral, one would look for injury to the cells of the cochlea; if bilateral, the superior olive or some other part of the central nuclear mechanism might be involved or possibly the cortex of Heschl's gyrus. The lesions producing athetoid cerebral palsy, whether located in the globus pallidus, substantia nigra, or premotor area of the cortex, might well be caused by anoxia also, but not likely by any lesion common to the extrapyramidal motor pathways and the auditory paths, since the two are everywhere widely separated.

It must be emphasized that the pathologic evidence in this analysis is limited to gross microscopic and neuropathologic examination without concomitant cochlear studies. As far as can be determined, no such correlated studies of brain and temporal bones in kernicterus have been made anywhere. Until this is done, the localization of the auditory lesions in intracranial nuclei must be considered purely theoretic. It is to be hoped that this report will stimulate such studies elsewhere.

As far as can be determined, bilirubin deposits in the temporal bone have never been recorded. Dr. Stacy Guild, in a personal communication, informed me that he has not encountered bilirubin deposits in cochleas. Dr. Dorothy Wolff stated in a personal communication that she has not encountered bilirubin deposits in her temporal bone studies.

The present report is a preliminary one. Correlated temporal bone and brain studies on kernicterus patients are being started.

The importance of anoxia in this condition must not be ignored. Windle's experiments and Larsell's observations, mentioned above, reinforce Dublin's interpretation of the anoxic mechanism in the production of the kernicteric distribution. The correlation of anoxia with the vascular anatomy of the cochlea and the brain stem may be highly significant.

In not a single case of athetosis and deafness was the deafness total. If we were to assume the existence of cochlear rather than central lesions in the icteric state, it would not be likely that the icteric deposition would involve only small cochlear areas. In view of the gross magnitude of icteric lesions in the brain, it would be expected that a cochlear lesion would also be potentially great, at least in some cases. However, an icteric lesion in the auditory pathway would be more likely to produce a lesser hearing loss in view of the diversity and bilaterality of the pathways.

Accordingly, the deafness in kernicterus has tentatively and theoretically been localized in the nuclear regions of the brain stem.

IMMUNOLOGIC TECHNICS

Determinations of major blood groups were made by routine saline methods using commercial sera. Rh factors were determined by the test-tube saline agglutination technics with 2 per cent suspension of the red cells under investigation. Commercial Anti-C (Rh′), Anti-D (Rh°), Anti-E (Rh″), and Anti-c (Hr′) sera were used in all instances.

Tests for maternal sensitization were performed with the father's or affected patient's cells whenever major blood groups would not interfere. Otherwise, tests were performed with O-Cde/e, O-cDE/cDE, O-cde/cde cells. Complete and incomplete antibody tests were performed in saline by agglutination and indirect Coombs' (developing) technic.[1]

SUMMARY OF OBSERVATIONS

Otologic-Serologic Study of a Group of Spastics

A total of 101 cerebral palsied children were studied otologically. The distribution of these cases was as follows: athetosis, 41; spastics, 38; rigidity, 9; mixed, 11; tremors, 2.

Of the 41 athetoids studied, there were 28 with normal hearing and 13 with significant hearing impairments. Of the 13 athetoids with hearing impairments, 11 had definite histories of erythroblastosis. In all of these 11 cases there was sufficient serologic evidence to substantiate the diagnosis of erythroblastosis.

Erythroblastosis was not encountered in any of the other cerebral palsied children studied.

None of the nonathetoid cerebral palsy group showed perceptive hearing impairments.

Deafness Questionnaire Analysis

The mothers of over 1500 profoundly deafened children were asked to fill out a questionnaire dealing with several aspects of a deafness research program. Approximately 900 intelligently and fully answered questionnaires were subjected to a preliminary scrutiny. A number of questions devoted to the Rh factor were studied in connection with this problem. These 900 cases include within their scope many well-defined groups of etiologic factors, but no attempt was made to remove such groups from the preliminary analysis. Therefore, among the many causes of deafness represented in the cases reported were meningitides, encephalitides, and hereditary factors, including 166 cases of maternal rubella in the first trimester of pregnancy.

Inasmuch as this is a random serologic group drawn from the general population, an expectancy of erythroblastosis of 3 to 6 cases among the 900 cases represented might be assumed, since the present ratio is considered to be 1 erythroblastotic to 150 to 250 births. It is quite striking, therefore, to discover 28 cases which can be classified as "probable" and 8 cases which are "possible" erythroblastotics.

A "probable" answer on the questionnaire was typically one indicating that the child was born severely jaundiced, required one or more transfusions, or was the Rh positive child of an Rh negative mother and Rh positive father. Rh information was not known in many cases, but where not stated, other weighting factors were given consideration in the classification of "probable" cases. Such factors as fetal mortality in other jaundiced siblings and the presence of spasticity in one or more surviving children were considered important weighting factors.

CASES OF ATHETOID CEREBRAL PALSIED CHILDREN WTH HEARING IMPAIRMENTS AND ERYTHROBLASTOSIS

The following 8 case histories of cerebral palsied children with hearing impairments are presented as somewhat typical of the larger group studied. This does not represent the total number of cases studied.

Case 1. *R.M.L.*

History. This second sibling, 4 years old, has an 11 year old normal sister. The mother was unable to conceive for seven years following the birth of the first child. There are no other children. There were no miscarriages.

Following an uneventful pregnancy, the patient was born definitely jaundiced and anemic. He required four transfusions following birth. The jaundice persisted until the age of 3 months. Shortly after birth, it was noticed that he had definite athetoid spasticity. Because of difficulty in speech, his parents suspected a hearing loss when he was about 18 months old. He still has no speech development of significance, and he shows definite hearing loss on subjective and audiometric testing.

Physical examination. There were no significant abnormalities in the ears, nose or throat.

Audiometry revealed a bilateral perceptive deafness with a loss of 50 db. in the speech range on each side (fig. 1). The curve is somewhat symmetric bilaterally.

Vestibular examination revealed a normal response to caloric stimulation in each ear.

Serologic study. Child—Rh positive; mother—Rh negative; father—Rh positive; older sibling—Rh positive. Antibody study on the mother: agglutinin titer negative. Moderate positive indirect developing (Coombs) test.

Diagnosis. Bilateral perceptive deafness of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 2. *G.W.*

History. This 10 year old boy is a second sibling. There is a perfectly normal older sibling, a boy aged 14 years. The patient had a normal delivery, following a normal pregnancy. He was jaundiced and anemic at birth, however. Spasticity quickly followed the jaundice. His life had a stormy course until the age of 2 months. Because of poor speech, the parents suspected a hearing defect which was first recognized at the age of 2 years. The child has an athetoid type of spasticity and is at present in a school for spastic children.

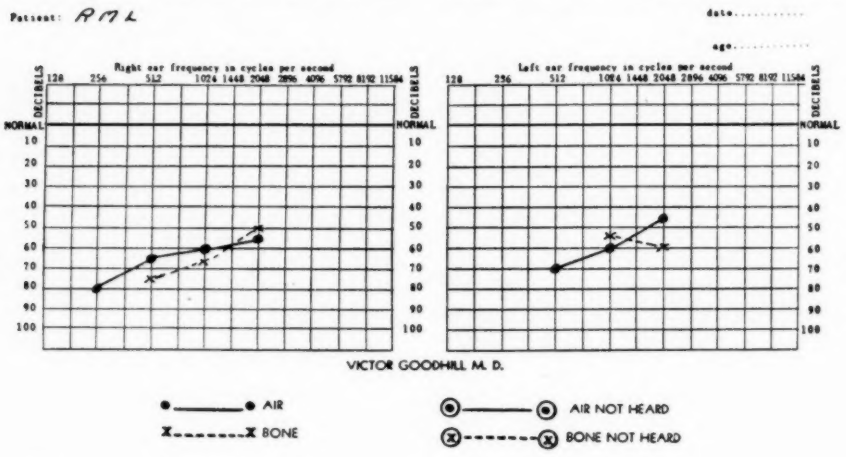

FIGURE 1.

Physical examination. There are no significant abnormalities in the ears, nose or throat.

Audiometry revealed a bilateral perceptive defect of approximately 50 db. in the speech range (fig. 2).

Serologic studies revealed that the father is Rh positive, the mother Rh negative, and the patient Rh positive. Antibody studies on the mother revealed a weakly positive indirect developing or Coombs test in 1949 when the patient was 10 years old.

Diagnosis. Bilateral perceptive deafness of nuclear origin due to kernicterus following erythroblastosis fetalis.

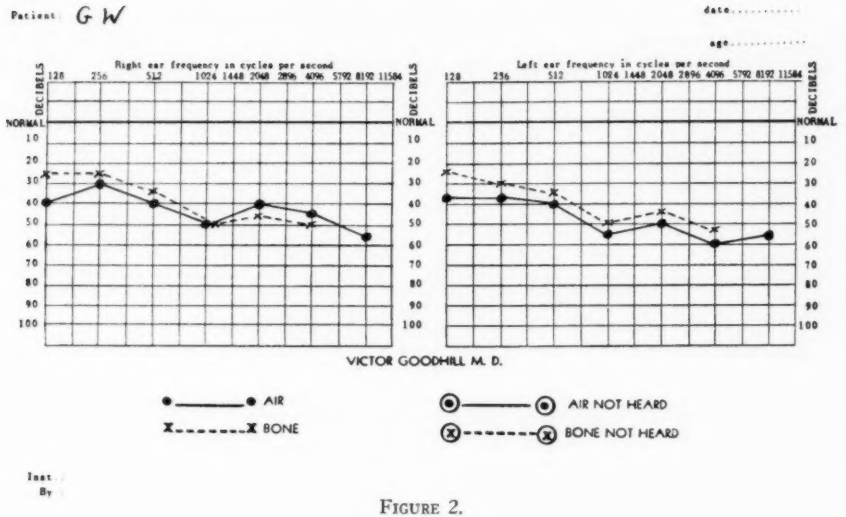

FIGURE 2.

Case 3. C.C.

History. This 9½ year old boy is the second child of a family of five children. The first child was perfectly normal. The birth history was negative. He was immediately jaundiced at birth, however. He was transfused once. Athetoid spasticity was noticed shortly afterward. There are two other children who are normal. A younger brother is also a spastic.

Physical examination. There were no significant physical abnormalities in the ears, nose or throat.

Audiometry revealed a bilateral dip at 2000 and 4000 cycles, perceptive in type (fig. 3).

Serologic Study. Father—Rh positive; mother—Rh negative; child—Rh positive. Indirect developing test (Coombs) on the mother was strongly positive for antibodies.

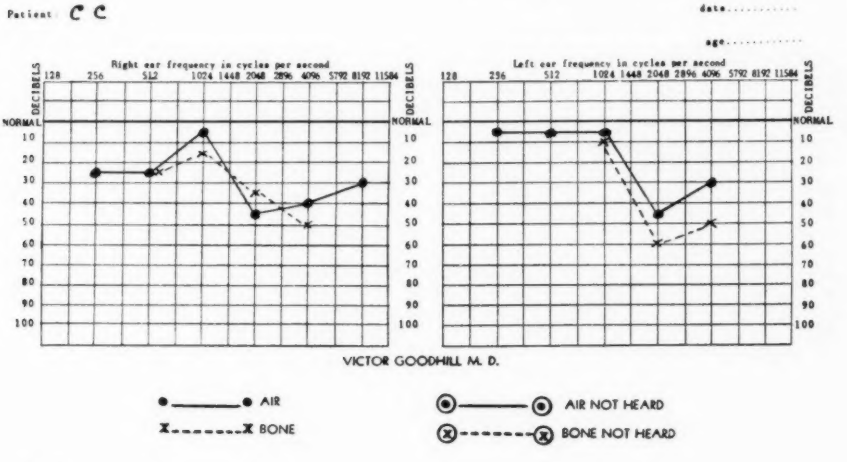

FIGURE 3.

Diagnosis: Bilateral high tone perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 4. *T.H.*

History. This is a 13 year old boy. There is one younger brother aged 4; no miscarriages. Birth history was normal except for the fact that the child developed jaundice one day after birth. This became progressively more intense and he was transfused several times. On the tenth day, postnatal, his neurologic manifestations revealed spasticity and episthotonus and a peculiar cry. A diagnosis of clinical kernicterus was then made. He had sev-

born sister nine years ago, of unknown etiology. The birth history was normal except for the fact that he developed jaundice within the first twenty-four hours and was transfused a number of times. Several months later a diagnosis of non-tension athetosis with mild quadriplegia was made. In view of a speech defect, audiometric examination was performed and revealed a hearing loss.

Physical examination. No significant abnormalities in the ears, nose or throat.

Audiometric examination revealed a bilateral perceptive deafness of approximately 45 db. in each ear (fig. 5).

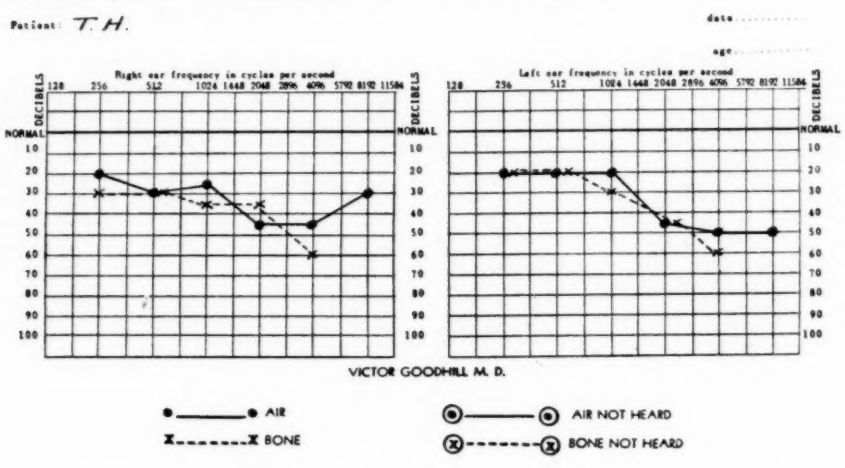

FIGURE 4.

eral convulsions following this episode and he was transfused again a month later.

Physical examination. No significant abnormalities in the ears, nose or throat. The child has an athetoid quadriplegia.

Audiometric examination revealed a bilateral moderate perceptive impairment, greater for the high tones (fig. 4).

Serologic study. Mother—Rh negative; father—Rh positive; child—Rh positive. Antibody test on mother revealed a positive weak reaction with indirect developing test.

Diagnosis. Bilateral high tone perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 5. *P.C.*

History. This 7 year old boy has a perfectly normal brother, aged 16. There was a still-

Serologic status. Father—Rh positive; mother—Rh negative; patient—Rh positive Antibody test on the mother revealed a strongly positive test by the indirect developing test method.

Diagnosis. Bilateral perceptive deafness of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 6. *P.D.*

History. This is a 4 year old boy. There were two previous pregnancies, both males; both died at the age of 4 days. Nothing else is known of their birth histories. This child was jaundiced at birth. Shortly following birth, an athetoid quadriplegia developed.

Physical findings. No significant abnormalities in the ears, nose, or throat with the exception of a small amount of lymphoid tissue

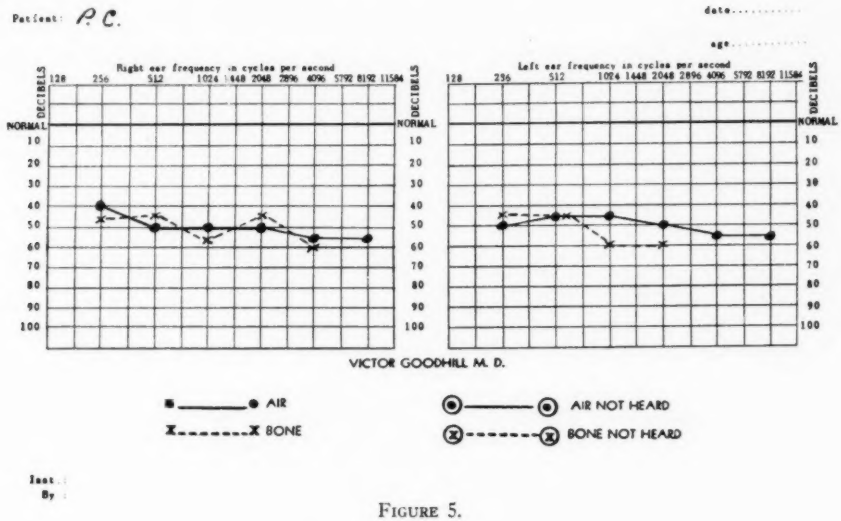

FIGURE 5.

in the nasopharynx, not in the tubal areas.

Audiometric examination revealed a bilateral perceptive impairment of approximately 40 db. (fig. 6).

Serologic examination. Father—Rh positive; mother—Rh negative; patient—Rh positive. Indirect developing test on the mother's blood revealed a moderately positive reaction against the father's and brother's cells. Conglutination test was positive against the father's cells.

Diagnosis. Bilateral perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 7. S.C.C.

History: The birth of this 11 year old girl followed a second pregnancy. (The first child was stillborn at seven months following a

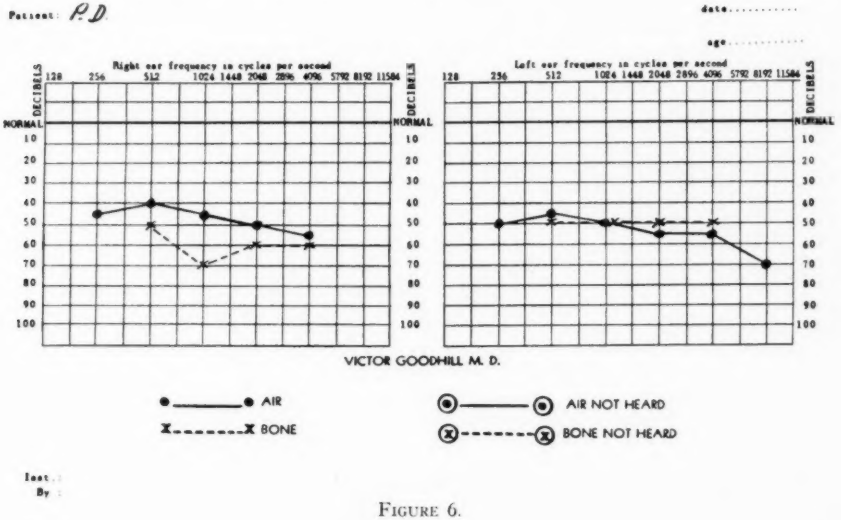

FIGURE 6.

placenta previa. The mother was transfused after the first pregnancy with her brother's blood. He was Rh positive.) The patient was severely jaundiced for approximately six weeks following birth. She had four transfusions and four spinal punctures; and a diagnosis of brain hemorrhage was made. She has since developed spastic findings and has been diagnosed as an athetoid quadriplegia with mild speech, hearing and visual impairments.

Physical findings. There are no significant abnormalities in the ears, nose or throat. Tonsils have been cleanly removed. The nasopharynx is negative.

Audiometric examination revealed a bilateral perceptive type of hearing impairment (fig. 7).

Serologic status. Father — Rh positive; mother—Rh negative; child—Rh positive. Maternal antibody determination — developing test positive against three types of cells.

Diagnosis. Bilateral high tone perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 8. A.A.

History. This 9 year old boy is the fourth child. The first child is living and well; the second pregnancy miscarried; the third child died of jaundice at the age of 3 days. The birth of the patient was at term following the fourth pregnancy. Jaundice appeared twelve hours after birth. He was transfused four times. Shortly after infancy, a diagnosis of athetoid quadriplegia with mild hearing loss was made.

Physical findings. No significant abnormalities in the ears, nose or throat.

Vestibular tests by rotary stimulation revealed no abnormalities.

Audiometric examination revealed a bilateral perceptive deafness of approximately 50 db. (fig. 8).

Serologic status. Father — Rh positive; mother—Rh negative; patient—Rh positive. Mother showed a weak positive reaction to the indirect developing test (Coombs).

Diagnosis. Bilateral high tone perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Discussion

It should be noted that each of the 8 cases of cerebral palsy was of the athetoid type and in each there was a bilateral perceptive hearing impairment. In no case was there complete deafness in either one or the other ear. In almost all of the cases the audiometric pattern was bilaterally symmetric.

TYPICAL CASES OF ERYTHROBLASTOSIS WITH IMPAIRED HEARING WITHOUT CEREBRAL PALSY

The following 3 case reports are not submitted for statistical study. They are

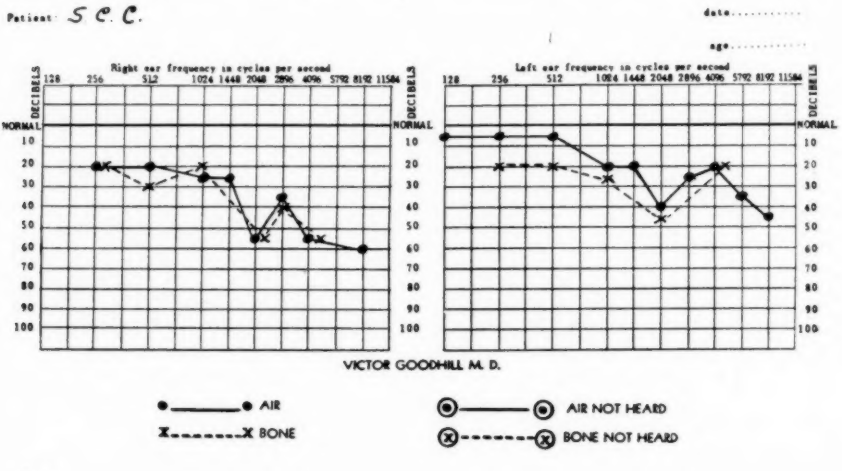

FIGURE 7.

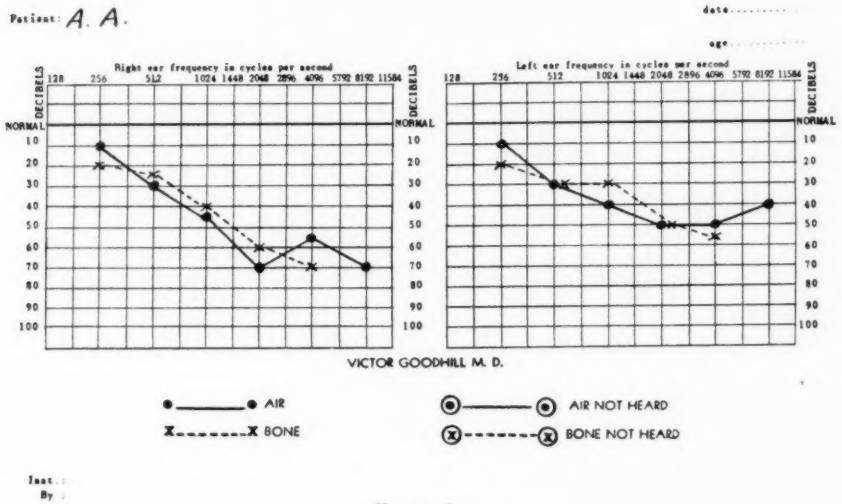
Patient: A. A.
date
age
Right ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100
VICTOR GOODHILL M. D.
AIR
BONE
AIR NOT HEARD
BONE NOT HEARD
Inst.:
By :

FIGURE 8.

simply random cases from a group of perceptive deafened children who gave histories and serologic findings consistent with the presumptive diagnosis of hemolytic disease of the newborn or erythroblastosis fetalis. These cases are significantly free of spasticity or athetosis.

Case 9. R.F.

History. This 14 year old girl is a second sibling. The first child died at birth with a defect in the spine and skull. The patient was born with subcutaneous hemorrhages but with no significant jaundice. She was found to be markedly anemic and was transfused twice. The mother developed a marked anemia and was ill for a long time. The third child was born with a cleft palate and hydrocephalus and died at the age of 2 years. A fourth child is alive now with a harelip and cleft palate at the age of 6 years. The patient was brought in because a loss of hearing had been noticed recently in school.

Physical examination. No significant physical findings with the exception of mild allergic rhinitis.

Audiometric examination revealed a completely deaf right ear and a moderate perceptive impairment in the left ear (fig. 9).

Vestibular tests revealed normal responses in both ears to caloric stimulation. There are no other significant findings.

Serologic status. Father — Rh positive; mother—Rh negative; patient—Rh positive. Mother has a weakly positive indirect developing test for Rh antibodies.

Diagnosis. Right complete and left partial perceptive deafness, possibly of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 10. E.A.

History. This 15 year old boy is a second sibling of Basque parents. The first child, a boy, is sixteen months older and is perfectly well. This child, the second, was born with a severe jaundice accompanied by some high fever. He slowly improved. The third child, a girl, was born two years later and died several minutes following birth.

The patient was somewhat ataxic between the ages of 1 and 5 but never had any spasticity. At the present time, he has no spastic findings. He has been known to be hard of hearing since birth. He has had difficulty in speech throughout his school career.

Physical examination. No significant ear, nose or throat abnormalities.

Vestibular tests revealed normal reaction to caloric stimulation on each side.

Audiometric examination revealed bilateral perceptive deafness (fig. 10).

Serologic study. Father—Rh unknown; mother—Rh negative; patient—Rh positive. The mother has a weakly positive indirect developing test for Rh antibodies.

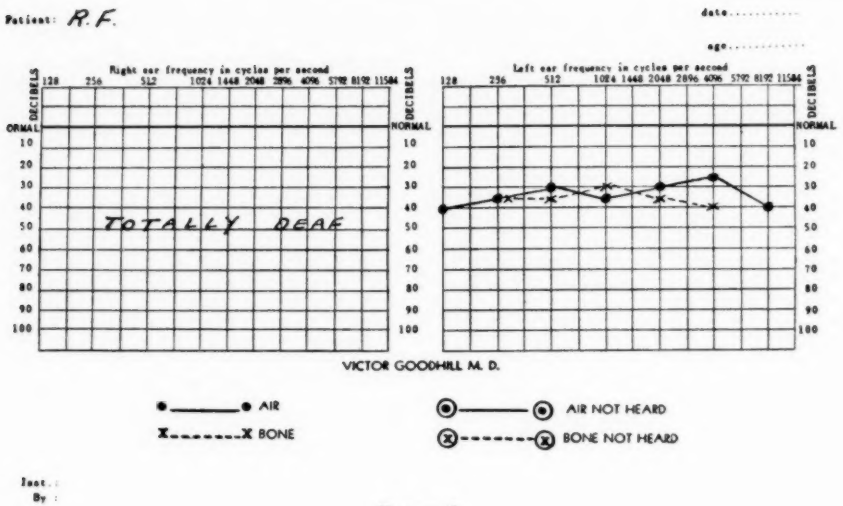

FIGURE 9.

Diagnosis. Bilateral high tone perceptive hearing impairment of nuclear origin due to kernicterus following erythroblastosis fetalis.

Case 11. *J.S.*

History. This 17 year old boy was first examined on June 18, 1947. He has had a known hearing impairment since early childhood, with no known cause.

The patient's father is a physician who stated that he, the father, was Rh positive and the mother was Rh negative. The patient was the first child, was born prematurely at seven months, and was jaundiced. He received one transfusion and then appeared to be well. He has never had any signs of spasticity or athetosis. The second and third children are normal. The fourth child died of typical erythroblastosis.

Physical examination. Physical findings were entirely negative. There were no abnormalities in the ears, nose or throat.

Caloric vestibular test revealed normal function in each labyrinth.

Audiograms revealed a bilateral perceptive impairment with a straight line sloping loss for the high frequencies (fig. 11).

Diagnosis. Bilateral perceptive deafness of nuclear origin due to kernicterus following erythroblastosis.

CONCLUSIONS

1. A clinical association of moderate bilateral perceptive deafness, athetoid cerebral palsy and history of erythroblastosis has been observed.

2. Kernicterus is the cause of the athetosis in the above group. It is probably related to the deafness as well.

3. The incidence of erythroblastosis in a large sampling of perceptively deafened children appears to be significantly *higher* than would be expected in the general population.

4. Evidence exists to localize the deafness resulting from erythroblastotic kernicterus in the nuclear masses of the brain and brain stem.

5. The Rh factor thus begins to play a role as a potential cause of some cases of infantile "nerve deafness."

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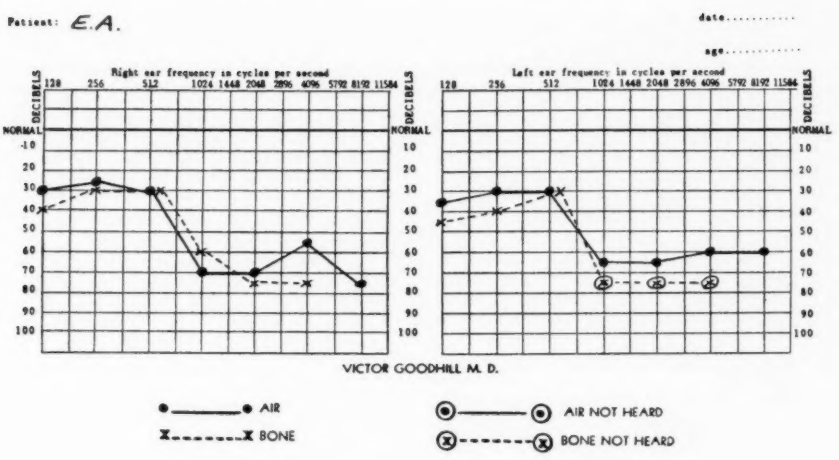

FIGURE 10.

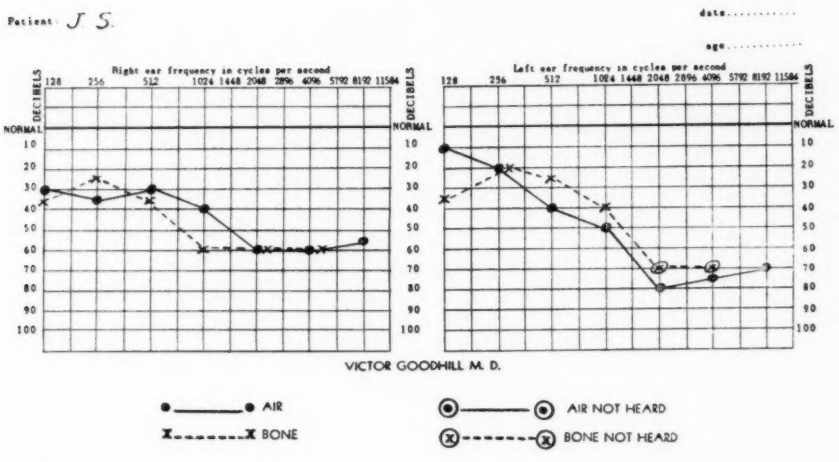

FIGURE 11.

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DISCUSSION

JAMES L. WILSON, M.D., Ann Arbor, Mich.: Dr. Goodhill has presented circumstantial evidence and data, as he says, not yet subjected to statistical analysis, which nevertheless is very convincing evidence that the disease erythroblastosis can cause deafness of central origin. I think he is right, not because I have been clever enough to detect this type of deafness with all the complex clinical symptoms that these children present, but because I think it is entirely logical from what we now know about the disease, that such a form of deafness and damage to the brain should occur.

I think certainly he is to be congratulated for this first analysis of such cases and his very careful and shrewd study. This disease complex has been a rapidly developing one in the last few years, as you know, and, as usually happens in these cases, a great many dogmatic statements have been made and intensive theorizing has been indulged in. We still do it. I should like to be dogmatic and to emphasize that we need to know a great deal more about the disease; that we don't yet know how to make dependable prognoses; that we certainly do not know how to treat these children dependably, and that we cannot prevent this injury to them.

The names that have been used to describe the disease, I think, also describe our confusion. The first one, icterus gravis, describes it probably about as well as any, in spite of the fact that we see this disease now without severe jaundice. The term "erythroblastosis" itself is simply a description of certain morphologic findings, but we have seen this disease complex without erythroblastosis, at least in the peripheral blood. "Hemolytic disease of the newborn" is another tempting label because the outstanding symptom of the disease is anemia resulting from hemolysis. But again this disease occurs without severe anemia in some cases. We have no good term, and we cannot struggle with complex labels such as reciprocal-sensitization-between-mother-and-baby-due-to-the-rhesus-factor disease, so it is good enough to call it erythroblastosis.

It was early thought that this disease complex caused its damage only by bringing about destruction of the sensitized red cells of the baby and that, if they could be removed and replaced with nonsensitized cells, we would have a cure. This theory was based on the assumption that erythrocytes were the only part of the body sensitized. Next it was thought that if we could replace all the red blood cells and plasma, too, we would have a cure. I think that is wrong and it is pretty evident now that this pathologic process, this sensitization process, involves tissues other than the blood tissues.

We have the picture, to which Dr. Goodhill referred, of the aplastic anemia of the newborn in which the bone marrow simply is aplastic and cannot make more red cells. Unless you want to assume such a vague etiologic concept as exhaustion of the bone marrow from overwork, for which we have no proof whatever, then we have to think specifically of the bone marrow as damaged—and the bone marrow is more than just peripheral red blood cells. It is probable that the liver also is specifically damaged, and we certainly have lots of evidence that the brain is damaged.

I regret that the term "kernicterus" has been used so much. It seems to me it is a little misleading because I like to think of it as only a pathologic term, one for the path-

ologist to use because he finds staining of basal ganglion. Actually, there is not a 100 per cent correlation between staining of the patient's ganglia and the existence of a damaged brain. Moreover, we find clinical evidence of far more widespread brain damage than could be explained by the distribution of the staining abnormality of the brain. I think that in our present state of knowledge it is safe to consider kernicterus as an incidental pathologic finding rather than an important step in pathogenesis, and that a more subtle cause of widespread damage to the brain occurs.

Children can be seen with many evidences of brain damage which presumably result from this disease, but we are still at a loss to identify the last common factor that causes this brain damage. Can we ever be certain that a damaged brain cell is the sole result of specific and direct damage from a mechanism such as erythroblastosis and not a secondary effect of generalized anoxia which occurs very frequently in this disease in the newborn, or of edema? It is exceedingly hard to separate all these factors, but I don't think we need hesitate to believe what Dr. Goodhill is pointing out—that central deafness of nuclear origin can be a direct result of erythroblastosis.

STACY R. GUILD, Ph.D., Baltimore, Md.: Dr. Goodhill's paper merits high praise, both for its factual content and for the intriguing series of new problems it raises. I am sure it will stimulate many investigations besides the ones he himself already has begun.

No one suspected, when the Rh factor of human blood was discovered nine years ago, that it might be associated with deafness except incidentally. Dr. Goodhill's evidence shows that without question a causal relationship does exist between Rh factor incompatibility, so-called, and numerous cases of congenital deafness. It is a fascinating story and an excellent example of how unpredictable are the applications that may be made of a discovery.

Furthermore, it should be noted that Dr. Goodhill's keen observations add another item to the list of systemic diseases that affect the field of the specialty of otology. It may not help or cheer the afflicted patients to know that the cause of the impaired hearing they have is an Rh factor incompatibility, but it certainly will be a satisfaction to otologists to be able to remove another group of cases from the classification "congenital deafness, cause unknown."

If, as now seems probable, kernicteric or other lesions of some part of the auditory pathways prove to be the immediate cause of the impaired hearing in persons such as Dr. Goodhill has described, treatment will be of no avail and the keynote of the attack on this form of deafness must be prevention. Many obstetricians, pediatricians, internists, laboratory scientists and others are actively studying the constitutional and physiologic factors that underlie the systemic disease. It is to be hoped that their investigations will lead not only to the discovery of better methods to keep such infants alive but also to the discovery of how to prevent the development of functional disabilities, including deafness, in the survivors. Certainly the prevention of nerve deafness due to kernicterus or to other lesions associated with erythroblastosis does not fall primarily in the province of the otologist, any more than does the prevention of deafness from epidemic meningitis or from diphtheria.

Again I congratulate Dr. Goodhill on his extremely interesting and stimulating contribution to knowledge.

Symposium: Pure Tone and Speech Testing

THE USE OF SPEECH TESTS FOR THE EVALUATION OF CLINICAL PROCEDURES

S. RICHARD SILVERMAN, PH.D.
ST. LOUIS, MO.

IN the past decade interest in clinical auditory tests has been greatly stimulated by a wide, albeit not wholly unrelated, variety of factors. The more significant among these are the development of electroacoustic instruments which make possible increasingly refined measurements, the growing interest in the fenestration operation, the expanding consumer acceptance of improved hearing aids, the evolution of promising technics of research in psychoacoustics and finally, but none the less stimulating, the encouraging co-operative attack by workers from allied fields on the problems presented by aural rehabilitation.

From the point of view of the clinical technics which they employ, auditory tests fall into three broad categories: (1) tuning forks, (2) pure tone audiometry, and (3) articulation tests.* Although the evolution of each of these tests overlaps that of the others somewhat in time, the order in which they are stated represents basically the sequence of their refined development. The basic principles of articulation testing have not been implemented universally in routine otologic practice, probably because of the recency of their emergence, the lack of standardization of operating technics and the unavailability of instrumentation. It is the purpose of this paper, therefore, to deal with the principles underlying articulation tests and to suggest how the results of the tests may be applied to clinical situations.

From the Central Institute for the Deaf, St. Louis. This paper is an adaptation with revisions of an address delivered by the writer to the Section of Otology of the Royal Society of Medicine, London, May 6, 1949.
Presented at the meeting of the Committee for the Conservation of Hearing, Oct. 9, 1949, Chicago, Ill.
*Promising tests for young children suspected of deafness using conditioning and electrical technics are beyond the scope of this paper.

THE ARTICULATION FUNCTION

The articulation function is the basic concept involved in articulation testing. Essentially, it expresses the relationship between the percentage of speech units (syllables, words, sentences) heard correctly and the intensity of the speech at the ear of the listener. The notion of the articulation function grew out of the attempts by Fletcher and his associates[6] at the Bell Telephone Laboratories to devise methods of assessing the efficiency of telephone systems in transmitting speech. The use of speech as the test material was a logical procedure since the purpose of the telephone obviously is to transmit speech. An example of one of Fletcher's early articulation functions[7] is given in figure 1. It is seen that as the speech gets more intense the listener hears more syllables correctly. A simpler form of articulation function is shown in figure 2.[10] The ordinate shows the articulation score in percentage of words repeated correctly and the abscissa gives the relative intensity in decibels. In this case, a list of 50 phonetically balanced words (recog-

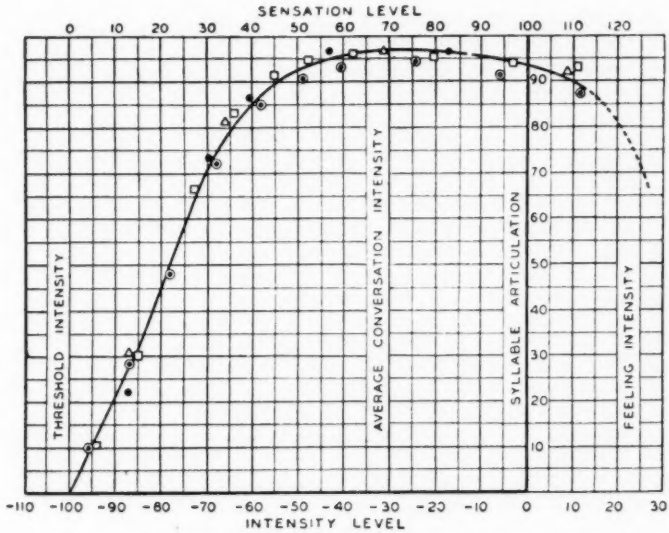
SENSATION LEVEL
0 10 20 30 40 50 60 70 80 90 100 110 120
THRESHOLD INTENSITY
AVERAGE CONVERSATION INTENSITY
SYLLABLE ARTICULATION
FEELING INTENSITY
90 80 70 60 50 40 30 20 10
-110 -100 -90 -80 -70 -60 -50 -40 -30 -20 -10 0 10 20 30
INTENSITY LEVEL

FIG. 1—Articulation vs. intensity of received speech. (From Fletcher.[7])

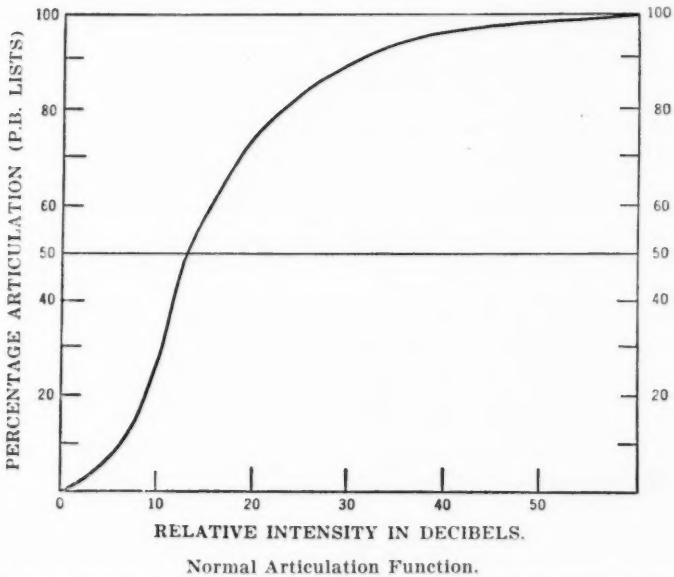
PERCENTAGE ARTICULATION (P.B. LISTS)
100 80 60 50 40 20
0 10 20 30 40 50
RELATIVE INTENSITY IN DECIBELS.
Normal Articulation Function.

FIG. 2—Articulation function for normal ear showing increase in percentage of words repeated correctly with increase in intensity. (From OSRD Report No. 3802.[10])

nized as proportionately representative of the phonetic elements of normal spoken English) is presented to the listener at enough levels, 10 db. apart, to complete the function. A subject, therefore, is credited with 2 per cent on the vertical axis for each word repeated correctly. Note also the manner in which the articulation score climbs as a function of intensity — especially the steepness of the slope and the 30 to 40 db. range over which almost maximum articulation is achieved.

It is apparent that articulation scores (and functions) are subject to the influence of many variables, and care should be taken to hold conditions as constant as possible in making tests except, of course, for the item under test, be it a communication system or an ear suspected of hearing impairment.

In figure 3 from Miller[9] we see, for example, how distance from lips to microphone may influence the percentage of word articulation.

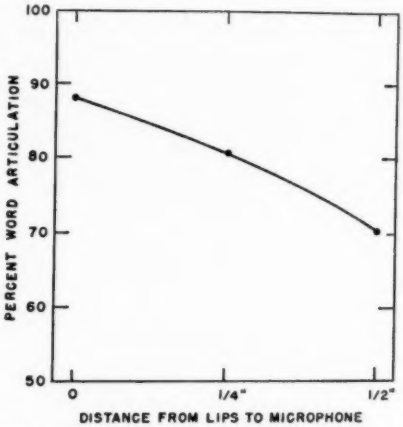

FIG. 3—The effect on communication of varying the distance of the microphone from the lips. The test with the announcer and crew of listeners in an airplane noise (115 db.). Each point is based upon the average of tests read by three announcers. (From Miller.[9])

DETERMINATION OF THE THRESHOLD OF HEARING BY ARTICULATION TESTS

It is well at the outset to clarify the concept of the threshold as it is derived from articulation tests. Three thresholds can be differentiated: (1) *the threshold of detectability* — defined as the point where the listener is just able to detect speech sounds about half the time and where he is not ordinarily able to identify any of the sounds themselves, (2) *the threshold of perceptibility* — defined as the point where the listener begins to perceive some words but where he can barely follow the gist of connected speech, and (3) *the threshold of intelligibility*—defined as the point where the listener understands half the material presented to him and where he can presumably follow without perceptible effort the gist of connected speech. For our purposes in the clinical context we shall need only the threshold of intelligibility.

In constructing a test to determine the threshold of intelligibility Hudgins et al[8] suggest that the test items should meet the following criteria: (1) *familiarity*—the vocabulary should be within the intellectual ken of the listener, (2) *phonetic dissimilarity*—fine discriminations (cowboy, plowboy) should not be necessary since no useful purpose is served by them in threshold tests, (3) *normal sampling of English speech sounds*—not essential for threshold testing but a reasonable sampling is desirable, and (4) *homogeneity with respect to basic audibility*—the ease with which test words are understood should be as equivalent as possible so that small numbers of items can be scrambled and the articulation function will rise steeply over a narrow range of intensity.

Hudgins and his co-workers at the Psycho-Acoustic Laboratory of Harvard University have developed a test which meets these criteria. It consists of two lists of 42 dissyllabic words of the

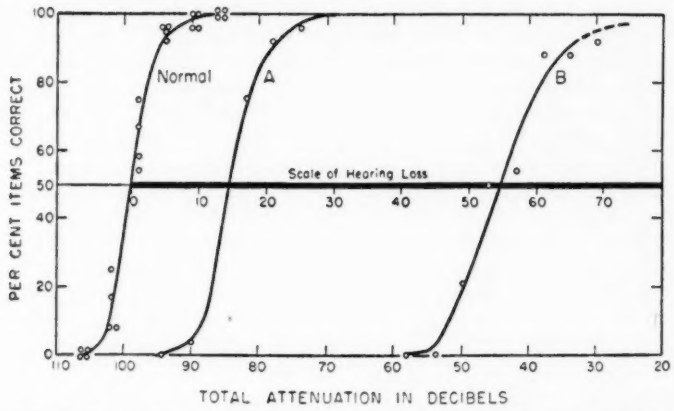

FIG. 4—The hearing loss for each of two subjects, A and B, is established in terms of the articulation function obtained from a group of normal listeners. Hearing loss is equivalent to the horizontal distance (in decibels) between the curve for the normal and that for the defective ear. Since the threshold is based on the normal score for 50 per cent correct, the horizontal line through the middle of the plot provides a scale of hearing loss. (From Hudgins.[8])

spondee stress pattern, that is, words such as *earthquake, hardware,* in which both syllables are accented equally. The subject hears six words at given levels progressively attenuated in 4 db. steps. The point at which he repeats correctly three of the words is the threshold of hearing (by our previous definition). Viewed from the standpoint of hearing impairment, the hearing loss can be defined as the additional number of decibels required by the impaired ear to hear the equivalent number of words (three). In figure 4 we see the hearing losses of two ears of different subjects. Subject A has a hearing loss of 14 db., and subject B has a loss of 55 db. In other words, subject A requires 14 db. and subject B, 55 db. more than the normal ear to hear the same number of words. The spondee test is usually referred to as "Test No. 9."

A sentence test (No. 12) based on the same principles also has been developed by the Psycho-Acoustic Laboratory.

The relationship of the percentage of word articulation and sentence intelligibility depends, of course, upon the type of words used and sentences used. A fairly typical curve of this relationship, plotted by Egan,[4] is given in figure 5. Hudgins et al[8] found that the threshold for the No. 12 (sentence intelligibility) test is about 4 db. above the threshold for No. 9 (spondees).

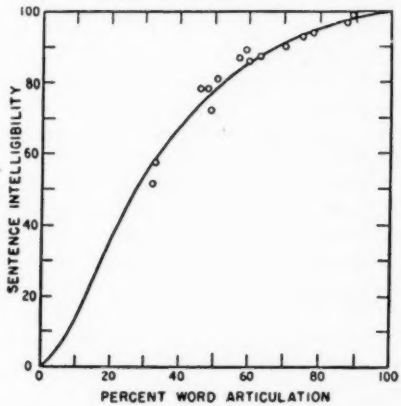

FIG. 5—Lists of words and lists of sentences were compared as to intelligibility under a wide variety of conditions. Each point on the graph represents one test of 100 words and one test of 50 sentences. The curve passing through the data was derived from results obtained at the Bell Telephone Laboratories. (From Egan.[4])

Another speech test (not using the concept of the articulation function) has been described by Falconer and Davis.[5] In this test the subject listens to a sample of recorded connected discourse and manipulates the attenuator until he just is able to get the gist of what is being said. His hearing loss is the difference between the number of decibels which he requires to reach his threshold of intelligibility for connected discourse and that required by the normal ear. This test has the advantages of economy of time and high face validity. The end point, on the other hand, is relatively not too definitive. However, Thurlow et al[12] found that hearing losses for speech determined by a variety of the types of tests described correlated so closely with one another that only one of them need be used in any clinical routine.

TESTING AUDITORY DISCRIMINATION

It is not only important to determine the faintest speech which an ear can hear (measured by threshold type tests described in previous section) but it is of great clinical significance (as we shall point out later) to know how the ear hears speech at any intensity, particularly above threshold. Clinicians are familiar with the patient who says, "I hear but I can't understand; I can hear better if you speak clearly rather than loudly." This patient's problem is not clearly delineated by the threshold test. Granted that a sound needs to be audible before it can be distinguished from the other sounds, the fact that it is audible does not necessarily guarantee that it can be recognized accurately. The ability to distinguish one sound from another when both are audible is called *discrimination* as distinguished from *sensitivity*, which refers to the faintest sound which the ear can hear. In addition to sensitivity, a high degree of discrimination is needed to discriminate *sin* from *thin* and *pit* from *pith*.

In constructing a test to measure the power of discrimination (or, clinically, the discrimination loss) Egan[4] suggests the following criteria: (1) *representation of fundamental speech sounds*—reasonable proportional representation in the test lists of the sounds that occur in everyday speech insures that the test measures what it sets out to measure, that is, how the ear copes with the task of discriminating speech which it is likely to have to discriminate in routine oral communication, (2) *types of test items*—words appear to be preferable to nonsense syllables which may require recording by phonetic symbols and to sentences which may afford contextual clues, and (3) *difficulty and reliability of test lists*—the test items must be so selected that the distribution of item difficulty in each list will make possible a sensitive measuring instrument. In other words, those items which under conditions of the tests are always recorded correctly, or are always missed, should be eliminated from the test lists. Of course, for both sensitivity and discrimination tests many equivalent lists should be available to cut down the learning factor when more than one test is necessary.

Egan[4] and his associates at the Psycho-Acoustic Laboratory have constructed a test which consists of 20 equivalent phonetically balanced lists of 50 words each. Figure 2 shows the articulation function which is derived from the P. B. (phonetically balanced) lists. The discrimination loss, measured along the vertical axis, would be the percentage of words that the ear could not discriminate at an intensity level sufficiently high above threshold to eliminate the factor of questionable sensitivity. In other words, no matter how loud the speech is made, the ear may still fail to discriminate all the test words.

A comparison of the shapes of the No. 9 and P.B. curves taken from Dav-

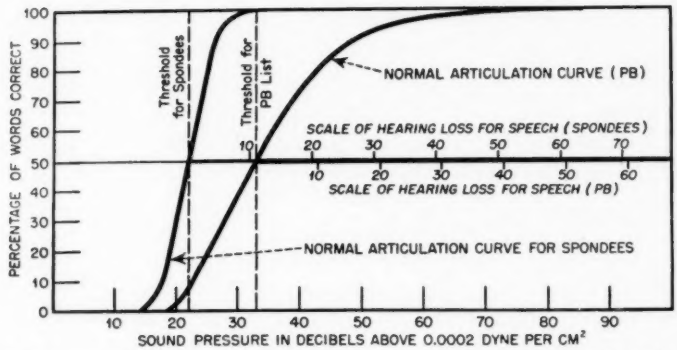

FIG. 6—The difference in slopes of two types of articulation curves. (From Davis.[1])

is[1] is shown in figure 6. Notice that the spondee curve rises steeply because the words are almost equally audible and intelligible while the P. B. curve rises more gradually because the words represent the broad range of difficulty encountered in everyday speech.

CLINICAL APPLICATIONS OF ARTICULATION TESTS

Articulation tests, properly applied, serve a useful purpose for the clinician who is confronted routinely by problems of diagnosis, prognosis and evaluation of medical and surgical procedures and hearing aids.

1. *Diagnosis and prognosis.* The otologist is called upon routinely to differentiate between deafness of the conductive and nerve types; in other words, he must assess cochlear function. The patient who is a candidate for fenestration surgery is a case in point. We can test the patient with the P. B. lists at high intensity levels (free from distortion) and if his articulation score increases proportionately with intensity he is likely to have adequate cochlear function. In essence, an attempt is made to accomplish prior to surgery what the surgery sets out to do, namely, to deliver sound to the analyzing mechanism in the inner ear by overriding or by-passing a conductive lesion. If the attempt to deliver sound over the electroacoustic system is successful as judged by the maximum articulation score, then surgery, intended to alter beneficially the transmission mechanism of the ear, should accomplish the same result.

In figure 7 we see the unsmoothed articulation functions taken from the clinical records of Walsh and Silverman.[13] Curve A is that of a patient who reaches a relatively high maximum articulation score if presented with speech at high intensity. This patient, therefore, has adequate cochlear reserve and would be a good candidate for surgery. Curve B represents a patient with mixed deafness, since his articulation score does not rise with intensity in the same manner as curve A. The conductive aspect of his deafness might be helped by fenestration, but his prognosis would be less favorable than that of the first patient. Note that in curve C increasing the intensity does not proportionately elevate the articulation score after a certain maximum has been reached. The curve for this patient shows that adequate cochlear function is not present. Surgery might provide for more sound to be delivered to the inner ear, but lack of adequate cochlear reserve would preclude reception beneficial to the patient.

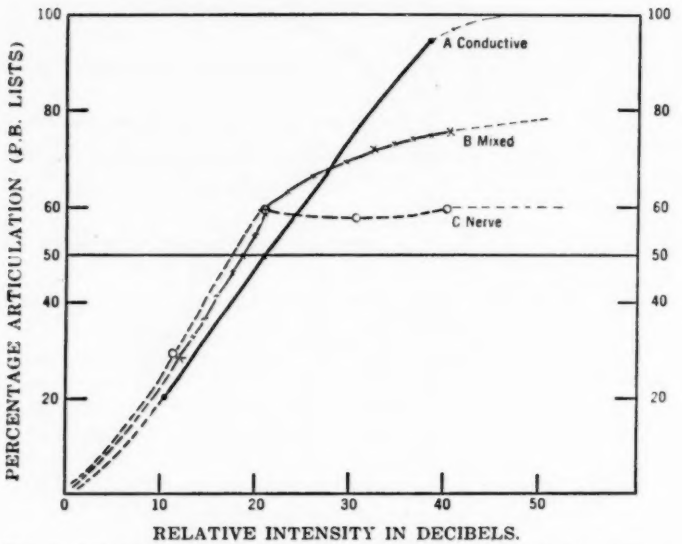

FIG. 7—Articulation curves for conductive, mixed and nerve type deafness. (From Walsh and Silverman.[13])

Walsh and Silverman point out further that the thresholds of the three curves are practically identical but the diagnosis and prognosis are based on auditory function above threshold; consequently, prime significance is attached to the *shape* of the articulation function and particularly to the *maximum articulation score*. The test would have obvious limitations in certain special types of perceptive deafness which, if suspected, must be explored carefully. For example, Dix, Hallpike and Hood[3] have suggested that the maximum articulation score might differ in cases of end organ lesions as against nerve fiber lesions.

2. *Evaluation and the concept of social adequacy.* In order to evaluate therapeutic procedures and hearing aids practically, we generally need to know to what extent we have improved the patient's ability to cope with the everyday dynamics of oral communication.

To this end Walsh and Silverman[13] suggested the concept of the *social adequacy index for hearing* (SAI) which essentially is the average percentage of P. B. words that would be correctly understood by the patient at the faint, conversational and loud speech levels. In other words, just as we are accustomed to averaging hearing loss at critical frequencies, we average ability to hear speech at critical intensities, in this case determined by social criteria. The threshold measure is not in itself adequate to predict a patient's performance since we have seen that ability to discriminate speech can vary widely above threshold in patients who may have the same threshold scores. The SAI, on the other hand, evaluates our remedial measures in terms of shifts both in sensitivity and discrimination. It tells us, for example, that the patient with poor discrimination is not helped by shouting, as we must in a noisy place, because an increase in intensity does not improve his power of discrimination. In a sense, the SAI accounts for hearing performance

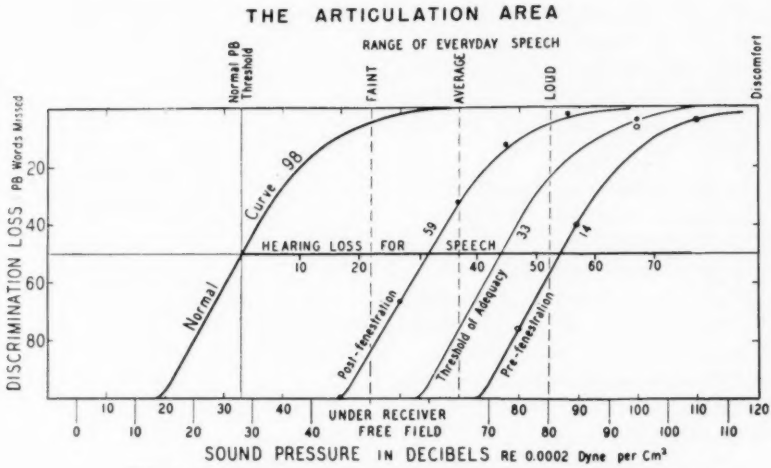

FIG. 8—The change in articulation function in the fenestrated ear.

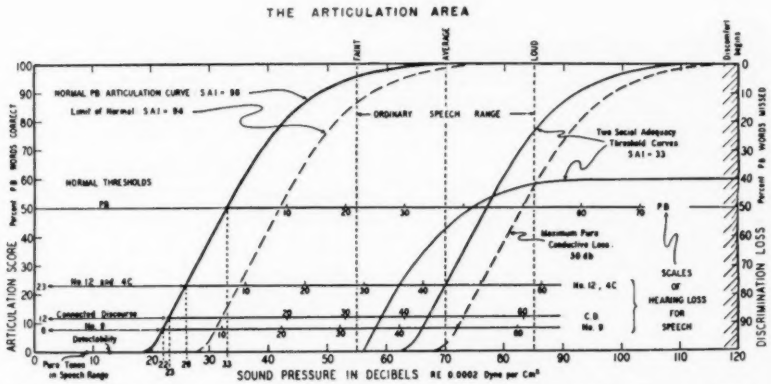

FIG. 9—Version 1, Feb. 1, 1948. These curves are based on the P.B. word lists of the Psycho-Acoustic Laboratory as recorded at Central Institute for the Deaf and used with the electro-acoustic installation there as of January 1948. The other thresholds are for the following recorded tests, using the same apparatus: (1) No. 9 (spondee words) and No. 12 (sentences)—Psycho-Acoustic Laboratory; (2) 4C (2-digit numbers)—Western Electric Co.; (3) Connected discourse (thresholds of intelligibility and detectability) newscast by Fulton Lewis, Jr.—Central Institute for the Deaf.

The Social Adequacy Index for Hearing is the average percentage of P.B. words that would be correctly understood by the patient at the three speech levels indicated by the dotted lines. (From Davis.[2])

in a dynamic acoustic environment. Of course, in cases where the discrimination is not expected to change perceptibly the shift in threshold will be adequate for evaluation. In figure 8 we see the articulation functions of an ear before and after the fenestration operation related to the critical threshold of adequacy and to the curve for the normal ear.

Davis[2] has elaborated the notion of the SAI and has brought into quantitative relationship the various articulation tests (fig. 9). He has also presented a table for the rapid calculation of the SAI.

Frequently, clinicians evaluate measures to improve hearing by soliciting a subjective judgment from the patient as to the status of his hearing. Silverman et al[11] studied the relationship in a series of 161 fenestration patients between their judgments of the status of their hearing and articulation tests performed under laboratory conditions. The patients were furnished a rating scale for various conditions under which they had to listen in everyday life (noisy places, audience situations, group conversation, etc.). Figures 10 and 11 show the relationships between patient judgments and actual measured losses and SAI scores respectively. Theoretically, we should expect linear relationships, that is, the greater the hearing loss (or the lower the SAI) the greater difficulty in hearing as judged by the patient. The plateau in both curves indicates that this is not so, probably because it is possible to compensate by the raised voice for losses of hearing up to 60 db. Note, too, the changes in judgments postoperatively. At any rate, patient judgment as a technic of evaluation should be employed with extreme caution. Table I, from Silverman et al,[11] indicates the numbers in terms of hearing loss and SAI which may be placed on certain critical social thresholds. Incidentally, this information may be useful to those who must deal with job classification and medicolegal problems.

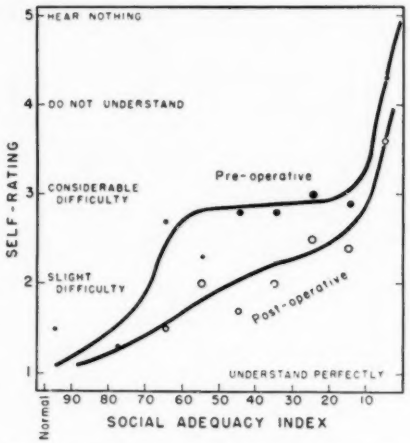

FIG. 10—The relationship between patient rating of hearing difficulty and the social adequacy index preoperative and postoperative. For some unexplained reason the patients seem to rate themselves more "accurately" after the operation. (From Silverman et al.[11])

EQUIPMENT

It is likely that simple, inexpensive assemblies of standardized equipment soon will be available and within reach of the practicing otologist, but even when this comes to pass precautions concerning characteristics and calibration must be observed. After all, articulation scores are relative scores contingent upon a variety of factors. Only relative statements can be made about them within a framework of accurately described equipment and conditions.

TABLE I

THRESHOLDS OF HEARING

THRESHOLD	LOSS FOR SPEECH DECIBELS	SOCIAL ADEQUACY INDEX
Normal	0	98
Limit of Normal	9	94
Social Difficulty Begins	30	67
Threshold of Social Adequacy	45	33
Limit of Compensation	60	10
Limit of Compensation with Hearing Aid	Still to be determined	

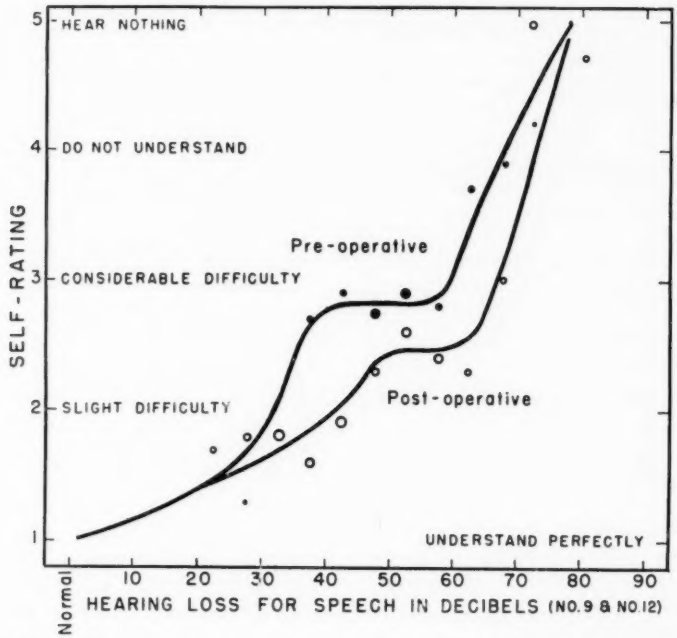

FIG. 11—The relationship between patient rating of hearing difficulty and hearing loss for speech preoperatively and postoperatively. (From Silverman et al.[11])

Finally, work in the field of articulation testing, at least in the present state of the art, should be viewed critically and, in some instances, experimentally. Ongoing research says that articulation tests need to be increasingly refined and validated and they need to be further streamlined for wider clinical application. Certainly this can be said about most clinical technics. Articulation tests are not intended to supplant orthodox clinical tests, but they are a valuable supplement and should hold a respectable place in the armamentarium of the clinical otologist because they are quantitative, reproducible, reasonably valid, and tend to objectivize the responses of the patient. After all, in articulation testing the patient either repeats the word correctly or he does not. He cannot get by with "maybe."

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THE CLINICAL APPLICATION OF BONE CONDUCTION AUDIOMETRY

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MANY otolaryngologists distrust bone conduction audiometry. Attempts to use it as a diagnostic tool in clinical practice have frequently been unsatisfactory and discouraging. It is true that some otologists have come to rely upon the technic, but they have encountered difficulty when comparing results. Generally speaking, bone conduction audiometry is considered a relatively uncertain method which serves merely as a diagnostic supplement to tuning-fork tests.

I

Four major reasons contribute to the current mistrust of bone conduction measurements.

In the first place, many workers doubt the reliability of thresholds obtained by this method. They feel that a high margin of error is introduced by such factors as differences in positioning of the vibrator and in the pressure with which it is applied. However, Carhart and Hayes[1] have shown that bone conduction thresholds can have good repeatability under clinical circumstances. They compared tests on five hundred ears. Statistically, thresholds for bone conduction were found to be superior in repeatability to thresholds for air conduction. It should be noted that all bone measurements were made with hearing-aid-type oscillators held in steel headbands. Positioning and pressure did not emerge as highly disturbing variables when this type of vibrator was used and when the ordinary precautions of clinical audiometry were maintained. Under such circumstances, a bone conduction curve may be interpreted with a margin of uncertainty no greater than the margin which must be assigned to the companion air conduction curve.

The second reason for distrusting bone conduction audiometry is more serious. It involves the calibration of audiometers. Manufacturers have disagreed as to the values to assign for normal thresholds. (To be sure, serious technical obstacles have been responsible. Nevertheless, the fact remains that we have not had clearly established standards.) The end result has been confusion when tests made with different audiometers have been compared. Furthermore, the situation has been aggravated because manufacturers have not always maintained as rigorous production and service standards for bone units as for air units. It is logical that persons attempting to use bone conduction should lose confidence in the method when faced with the uncertain validity which these factors represent.

The third reason for doubting the bone technic has arisen from inadequate

From the department of otolaryngology, Northwestern University Medical School.
Presented at the meeting of the Committee on the Conservation of Hearing, Oct. 9, 1949, Chicago, Ill.

testing conditions and imperfect office checks on calibration. Again, technical obstacles are numerous, but they are not serious if clearly understood. For one thing, even small amounts of room noise will mask the normal ear while it is being tested by bone conduction. In consequence, false thresholds will be obtained with patients having near-normal hearing. False thresholds will also appear in attempts to calibrate the bone unit with normal ears. Faulty calibration leads to incorrect positioning of the bone conduction curve on the audiogram chart. Whenever the error is large, the resultant air-bone relationships will be illogical in view of other diagnostic criteria. Quite naturally, the bone measurements are disregarded and the technic is distrusted.

Finally, bone conduction audiometry places special emphasis on controlled masking of the opposite ear. Unfortunately, the clinical application of controlled masking has been shrouded in confusion. Its practical use requires a calibrated noise whose effective masking is known. Most audiometers and special masking units have not been supplied with a noise which is so calibrated. Usually, therefore, the busy physician has been forced to work with an unknown—and frequently inadequate — quantity when he attempted to apply controlled masking in his pure tone audiometry. His bone conduction measurements have suffered most because masking of the opposite ear is more often critical in this kind of testing and because of threshold shifts which occur when some types of ear are occluded by the masking headphone.

II

The limitations just reviewed are not insurmountable. Adequate bone conduction audiometry has four special requirements.

In the first place, the kind of audiometry room which is acceptable depends upon the type of hearing one plans to test. If one wishes accurate bone thresholds on normal and near-normal listeners, a highly soundproofed chamber is essential. However, one may have to be satisfied with a room which, though relatively quiet, is not fully soundproof. Here adequate tests with bone conduction are impossible on normals, but one can still obtain useful measurements on hard-of-hearing patients. Stated differently, *any room which is adequate for air conduction testing of normal hearing is also adequate for testing the bone conduction of patients whose hearing loss is sufficient to constitute a social handicap.* For example, good bone measurements may be made on patients whose losses are severe enough to warrant consideration of fenestration surgery. Their hearing losses serve as protection against masking by mild ambient noise. Good testing by bone requires that one remember the limitations which a particular audiometry room imposes and that one restrict accordingly the types of patient given bone tests.

The second requirement is that the audiometer itself shall be an approved instrument which is functioning satisfactorily. General performance can be assured by checking it in the office and by returning the instrument periodically to the manufacturer for service. However, the user must at present make his own empirical calibration of its bone conduction system. Such calibration is easily accomplished. It requires a moderately good testing room and involves three steps.

1. The air condition circuit is first calibrated on ten or twelve young adults whose hearing is known to be normal. The average reading for this group is taken as the norm at each frequency.

2. Next, ten to twelve ears* having pure perceptive losses of intermediate severity are tested over the 256 to 4096 c.p.s. range, by both air and bone. Theoretically, such cases should yield superimposed air and bone curves. If the curves do not superimpose, appropriate corrections should be made in the values assigned to the bone readings. The method for making such corrections is illustrated in figure 1.

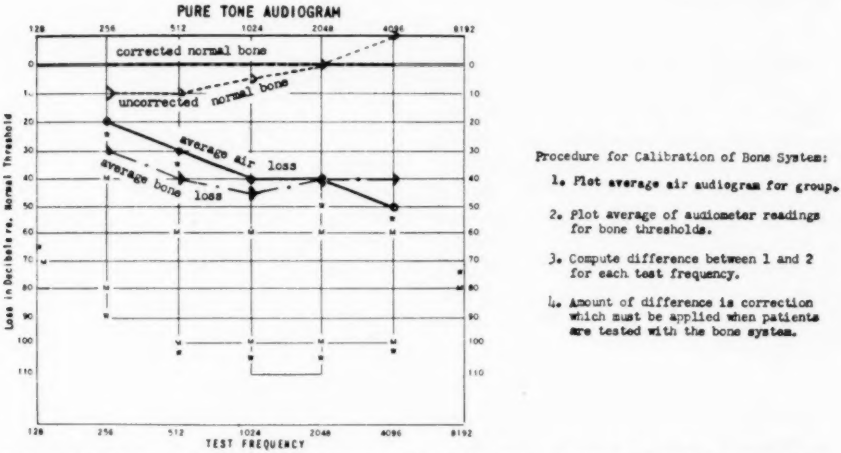
PURE TONE AUDIOGRAM
corrected normal bone
uncorrected normal bone
average air loss
average bone loss
Loss in Decibels re. Normal Threshold
TEST FREQUENCY
Procedure for Calibration of Bone System:
1. Plot average air audiogram for group.
2. Plot average of audiometer readings for bone thresholds.
3. Compute difference between 1 and 2 for each test frequency.
4. Amount of difference is correction which must be applied when patients are tested with the bone system.

FIG. 1—Method for calibrating bone conduction system with a group of cases with mild perceptive losses.

   a. The average air conduction curve for the group of perceptive losses is first plotted. (Audiometer readings are naturally corrected where necessary in order to conform to the calibration for air obtained in step 1.)
   b. Next, the averages of the *audiometer readings* for bone are plotted on the same chart.
   c. The amount, at each frequency, by which the two curves fail to agree represents the correction which must be applied to bone readings for that frequency.
3. The third step is to compute from these corrections the audiometer settings for bone which correspond to normal threshold. This last is done to the nearest five decibels.

Once the calibration just described has been completed, clinically acceptable measurements of bone conduction threshold may be obtained. The audiometrist merely corrects subsequent audiometric readings for bone by the amounts which the calibration data require. The procedure is simplified by pasting a correction sheet on the face of the audiometer. The audiometrist, when testing a patient, can then glance at the sheet and record the corrected threshold rather than the instrument reading. The resultant audiometric chart will show air-bone relationships which are more easily interpreted.

The basic advantage of the foregoing method for calibration is that it is not affected by mild room noise. In the writer's experience, after this type of empirical calibration, bone curves are obtained

*Each patient selected should have approximately equal loss in the two ears. Observing this precaution avoids complications and confusion. However, different patients need not have identical losses.

on hard-of-hearing patients which are credible in the light of other diagnostic criteria.

The reader will note that clinical audiograms offer a continual check on the adequacy of the calibration. Patients with pure perceptive losses should yield records which, within the limits of accuracy of clinical audiometry, show superimposition of the air and bone curves. When such records are not obtained, the calibration is incorrect and the whole procedure of calibration must be repeated.

Unfortunately, time limitations allow only mention of the last two requirements for adequate bone conduction testing. The third requirement is an effective system for controlled masking of the opposite ear. The fourth requirement is that the audiometrist be well trained and have good insight. The audiometrist must be capable of adapting her testing routines to the patient and to the details of the loss as they emerge during the test.

III

Whenever adequate bone conduction audiometry is available, results can be obtained which are highly useful in diagnosis. Lierle and Reger[4] have stated the basic premises.

They say (pp. 222-223):

I. If the air conduction acuity is below normal but bone conduction normal or possibly increased, the patient has a middle ear lesion.

II. If the patient has the same loss for both air and bone conduction sound, or possibly disproportionately decreased bone conduction, the loss is the result of an inner ear lesion. . . .

III. If the patient has a loss for both air and bone conducted sound, with a greater loss for air than for bone conduction, the impairment is due to a mixed lesion, indicating simultaneous middle and inner ear involvement.

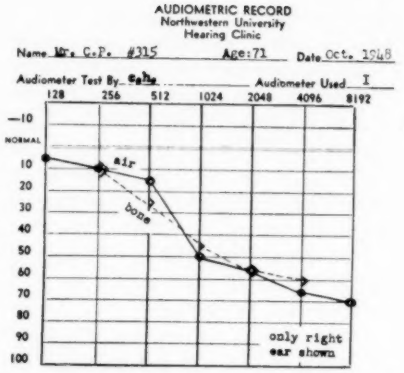

FIG. 2—Air and bone audiograms for right ear of elderly male with deafness due to presbycusis illustrate superimposition of curves within limits of accuracy of clinical audiometry.

When testing is properly controlled, routine audiograms conform to the foregoing rules within the limits of accuracy one may reasonably expect of clinical audiometry. The following examples will illustrate this point.*

Figure 2 shows the audiometric record on an ear with nerve deafness which was diagnosed as presbycusis. One is impressed by the manner in which the air and bone curves interweave. Within the limits of accuracy of clinical audiometry, the losses are equal by the two modalities. The case gives no audiometric evidence of a conductive component. This finding was confirmed by all the other diagnostic criteria.

Figure 3 illustrates another type of pure perceptive loss, namely, acoustic trauma due to exposure to machine-gun fire. Here, the outstanding feature is the sharp drop in acuity for high frequencies. The air and bone curves again interweave. The loss for bone is the same as the loss for air—as theory implies for a perceptive lesion.

*Diagnoses on the cases discussed within this paper were supplied by Dr. George E. Shambaugh, Jr., Dr. Arthur Juers, and Dr. Paul Campbell.

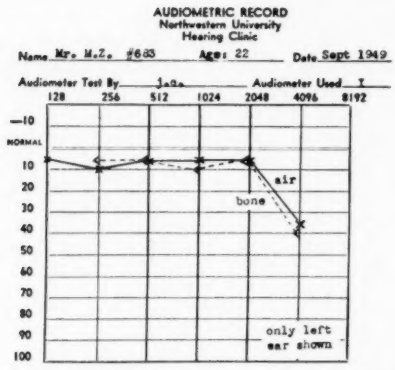

FIG. 3—Air and bone audiograms for left ear of young adult with high tone perceptive loss due to acoustic trauma as result of exposure to machine-gun fire. Illustrates superimposition of curves within limits of accuracy of clinical audiometry.

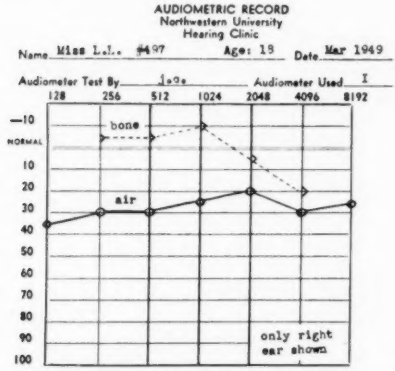

FIG. 4—Air and bone audiograms for right ear of teen-age female with conductive deafness due to chronic suppurative otitis media. Area between the air and bone curves represents the conductive component, within the limits of accuracy of clinical audiometry.

Figure 4 portrays the audiogram yielded by an ear with conductive deafness due to chronic suppurative otitis media. Here, the bone curve is above the normal threshold line except for a drop in the high frequency range, while the air curve is depressed an average of 28 decibels. The gap between the two curves represents the conductive component.

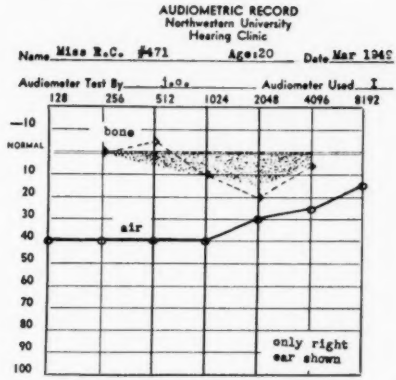

FIG. 5—Air and bone audiograms for right ear of young adult female having clinical otosclerosis with incomplete ankylosis of stapes but without inner ear involvement. Shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors.

A wide variety of additional examples could be given, but we shall limit the remainder of this discussion to some of the audiometric patterns which have been yielded by patients having clinical ostosclerosis.

IV

Experience in the Hearing Clinic at the Northwestern University Medical School has revealed a typical audiometric configuration which has frequently been found in clinical otosclerosis without cochlear damage. Figure 5 illustrates this configuration as encountered in a case with incomplete ankylosis of the stapes. One notes two features. First, the air conduction curve rises somewhat at higher frequencies. As will be pointed out later, the slope of the rise appears to be related to the stage in the progression of stapes fixation. Second, the bone conduction contour carries a shallow notch with its greatest depression at 2048 c.p.s.

The notch in the bone conduction curve is particularly provocative since it seems to imply a slight component of nerve loss. However, evidence is accum-

ulating which indicates that this notch is the result of purely mechanical factors. In other words, there seems to be a shift in sensitivity to bone stimuli which appears in connection with the process of stapedial fixation. This shift has been observed before fixation is complete. Its details have not been fully defined, but the shaded area in figure 5 represents the estimate as to its magnitude which the writer has made on the basis of the clinical evidence at his disposal.

It is still too early to have a final opinion on this point, and research is continuing. However, the clinical evidence is most provocative. Figure 6 presents the preoperative and postoperative audiograms for an ear subjected to fenestration surgery.* One notes that both the air and bone conduction curves show postoperative improvement. The bottom chart in the figure compares the two bone levels. The shaded area shown in this chart represents the writer's present

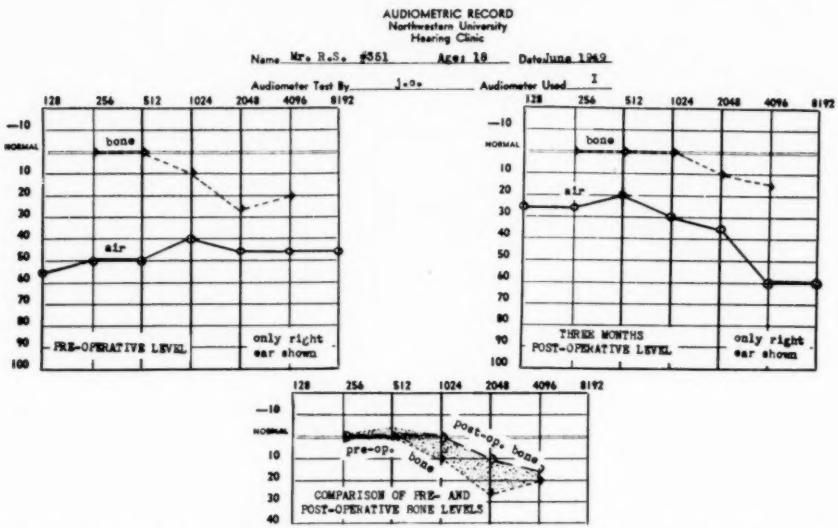

FIG. 6—Shift in bone sensitivity following fenestration surgery. Shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors. The postoperative bone curve shows improvement by an amount almost identical with the shaded area.

This evidence is of two types. For one thing, the notch repeatedly characterizes cases which in other respects appear free from cochlear damage. Secondly, Juers[3] has presented data which suggest that bone conduction is improved in the fenestrated ear. The writer and his associates have analyzed Juers' data statistically. The pattern of improvement is almost a direct reversal of the notch just mentioned.

estimate of the shift in bone sensitivity due to mechanical factors associated with stapedial fixation. One observes that the separation between the two bone curves corresponds very closely to the shaded area. This case offers one example of the clinical evidence which suggests to the writer that (1) fixation of the stapes modifies the mechanical

*As with all cases reported here, tests were conducted under the writer's supervision in the Hearing Clinic at Northwestern University.

constants of the inner ear, so that *its frequency response to skull-borne vibration is changed,* and (2) fenestration surgery restores the mechanical constants to approximately their earlier values, so that there is a reversal in acuity for bone conduction stimuli. It is possible that resistance factors (in the physicist's sense) in the inner ear are involved.

The foregoing hypothesis does not invalidate the clinical premise that the bone conduction curve gives an indication of nerve function. Changes in bone threshold due to mechanical factors are minor in degree. They are superimposed upon loss for bone conduction due to inner ear impairment. For example, where stapes fixation is associated with inner ear deafness, the notch due to mechanical factors is added to the abnormality in the bone curve resulting from the perceptive lesion (fig. 7). The bone curve may be considered a composite of a mechanically induced notch and an inner ear loss. The component representing the mechanically induced notch is indicated by the shaded area. The gap between the shaded area and the bone curve represents the component of inner ear deafness. Notice that when one corrects the air curve by an amount equal to this component, the resulting curve for the conductive component lies at a level and has a slope which is theoretically credible for a case of stapes fixation without cochlear damage. This case exemplifies superimposition of conductive and perceptive components in the probable presence of a small shift in bone sensitivity due to mechanical factors.

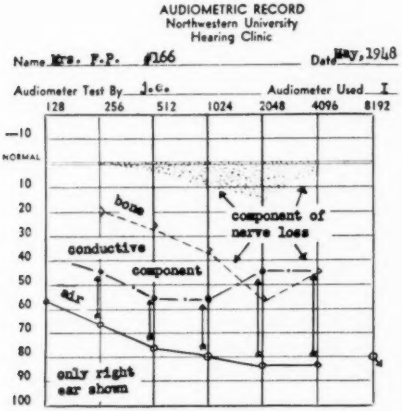

FIG. 7—Air and bone audiograms for right ear of adult female having clinical otosclerosis with secondary nerve involvement. Conductive component is air curve minus component of nerve loss (i.e., gap between bone curve and shaded area). Shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors.

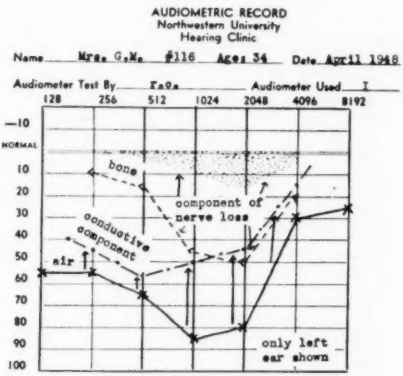

FIG. 8—Air and bone audiograms for left ear of adult female having clinical otosclerosis with secondary nerve involvement. Conductive component is air curve minus component of nerve loss (i.e., gap between bone curve and shaded area). Shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors.

A more dramatic example is shown in figure 8. The diagnosis in this case was otosclerosis with secondary nerve involvement. Notice the substantial parallelism in the troughs which appear on the air and bone curves. Again, the shaded area indicates the component of inner ear loss. This area may be subtracted from the bone conduction audiogram to yield the component of inner ear deafness. The conductive component may be considered as the difference between the air audiogram and the component of nerve loss.

Another interesting aspect of the audiometric contours characterizing clinical otosclerosis involves the impedance formulas discussed by Johansen[2] and others. We shall consider here only the shape of the gap separating the air and bone curves.

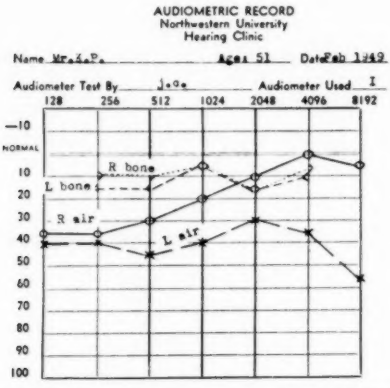

FIG. 9—Air and bone audiograms for adult male having unequal progress of clinical otosclerosis in the two ears. The air curve for the right ear (partial fixation of stapes) reveals loss for low frequencies. This curve is suggestive of increased stiffness. The air curve for the left ear (stapes fixation) shows additional loss involving high frequencies. The additional loss suggests increased mass, probably because the stapes is mechanically loaded with the adjacent bony tissue.

Figure 9 presents the pure tone thresholds for a case of clinical otosclerosis with unequal progress of the disease in the two ears. Notice that both ears show some notching of the bone curve. Notice further that the air conduction curve for the right ear rises for higher frequencies and reaches the normal line at 4096 c.p.s. In other words, this ear has been affected primarily in the low frequencies. According to the impedance formulas, such a change should result from increased stiffness in the conductive mechanism. By contrast, the air conduction curve for the left ear is a composite of the configuration for the right plus a depression of hearing for higher frequencies. The impedance formulas account for an additional change of this type on the basis of a mass increase.

It would appear from such clinical evidence that the progression in stapes fixation occurring in otosclerosis is characterized first by increased stiffness and subsequently by increased mass. A sequence of this type is entirely logical. The initial stages of fixation do not fully preclude vibration but offer obstruction which would change the elasticity of the system. A stage must be reached, however, when fixation is extreme enough so that (from the mechanical point of view) the stapes becomes loaded by the adjacent temporal bone mass.

Proper audiometry can have great diagnostic importance when one is dealing with such a progression. It may encourage earlier and more definitive recognition of clinical otosclerosis. It may allow stages in the pathologic change to be specified with greater surety. If the time ever comes when medical therapy for otosclerosis is perfected, proper audiometry can encourage more effective therapy by aiding in early discovery of cases.

V

The foregoing discussion of clinical otosclerosis has been presented partly for its illustrative value. We have only begun to take advantage of the contribution to diagnosis which bone conduction audiometry can make. The first step has been to perfect the methodology. Measurements which are clinically adequate can now be obtained if proper precautions are observed. However, both technical problems and a widespread mistrust must be overcome before bone conduction audiometry becomes widely used. Furthermore, most of the finer diagnostic interpretations which air-bone relationships may eventually allow still await discovery. Already some of these finer implications appear to have emerged in the study of clinical oto-

sclerosis. Future research and clinical application will make bone conduction audiometry a diagnostic tool of increasing value to otology.

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PHYSIOLOGY AND PATHOLOGY OF SOUND CONDUCTION IN THE EAR

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In 1543 Vesalius published his famous masterpiece of anatomy, "De Corporis Humani Fabrica libri septem." This work contained important contributions to the morphology of the ear. About the function of the ear, however, Vesalius summed up his knowledge by writing:

"Nihil certum est de auditu sonituque percipiendo." "Nothing is certain about hearing and the perception of sound."

Four hundred and six years later, in the year 1949, Bast and Anson published a book on the temporal bone in which they point out that "the ear of all organs of sense perception is the best protected, the most delicate, structurally the most complicated, and functionally the least understood. New facts regarding its structure are still being found, pathology and dysfunction of the ear still baffle the otologist; and the conflicting interpretations as to hearing still belong to the realm of theory."

The disappointing and painfully slow progress of our knowledge on hearing is not due to lack of interest of otologists or biologists. For centuries scientists have labored on the problem of audition. The reason for our failure undoubtedly lies in the unusual difficulties in obtaining experimental information. A clear understanding of the physiology of hearing is more important than ever, because the otologist of today is confronted with problems quite different from those of twenty years ago. Two decades ago the treatment of inflammatory diseases was the main problem of the ear specialist. Today the most outstanding symbol of modern otology is the fenestration operation.

The surgical restoration of hearing not only requires the acquisition of surgical skill; it also necessitates a better knowledge of the physiology of hearing. It should be said here frankly and without reservations that our knowledge of the biophysics of the ear is inadequate. We cannot explain fully how the fenestrated ear hears. The operation is based on empirical knowledge that a fenestrated ear does hear. Additional operations for the restoration of hearing are being described and more will be attempted; therefore our ignorance of the biophysics of hearing is the more deplorable and intolerable.

The three main difficulties are as follows:

1. The inaccessibility of the ear. The most important parts of the ear are embedded deep inside the skull. The only visible structure is the functionally unimportant pinna and the lateral parts of the external auditory meatus. In a person with normal ears, the otologist can visualize the ear drum. Beyond that he cannot observe the ear under normal conditions. The ear drum is the "iron curtain" of the otologist.

2. The second difficulty for our understanding of the ear is the smallness of the structures. For this reason histologic examination of the ear, in spite of its difficulties and not withstanding its limitations, has been the method of study for the last one hundred years.

From the division of otolaryngology of the University of Chicago.
Presented at the meeting of the Committee on the Conservation of Hearing, Oct. 9, 1949, Chicago, Ill.

3. The third fundamental difficulty lies in the nature of sound. Acoustic oscillations are rapid phenomena. The human eye cannot see repetitive movements of sound frequency because the fusion frequency of the visual sense organ is lower than the frequency of the oscillations. When enumerating these difficulties, it becomes clear why the otologist during the last eight decades mostly chose, or was forced to choose, the microscopic approach. The microscope solves the first two difficulties, at least to a certain extent. Serial sections of the temporal bone make the remote inner ear accessible to the eye. The lenses of the microscope enlarge the structures to an extent that most important morphologic details can be described. It is hardly necessary to enumerate the achievements of the histologic studies of the ear. The morphologic studies of Corti, Hensen, Deiters, Claudius, Reissner, and others are commemorated by the fact that their names have been attached to some of the ear structures. On the debit side of the microscopic technic stands the time consuming and expensive quality of otologic histology. More serious yet is the fact that important aspects of audition can never be solved by microscopic tissue examinations.

The best method to learn more about the function of the ear seemed to be in direct observations of the ear during acoustic stimulation. The complex phenomena of the ear should be dissolved into their basic physical components. By this approach we can hope to learn more about the physiology and pathology of sound conduction in the ear.

In this paper the attempt is being made to acquaint you with our experimental method, which tries to utilize the good features of the microscope without accepting the disadvantages of histologic technic. This method can be compared with the biomicroscopy of the eye. Using proper illumination and lenses of varying magnifying powers, the structures of the ear are observed without having gone through the intricacies of histologic technic, such as fixation, dehydration, decalcification, sectioning and staining. By this method the structures are observed in the animal during life or in human cadaver ears soon after death. There is considerable experimental evidence available that the ossicles, ligaments, membranes and spaces of the ear do not change immediately after death in regard to their physical behavior. It is therefore possible to utilize fresh human temporal bones for the study of all the structures which, intra vitam, are inaccessible.

The problem of optical magnification was easily solved by conventional optical lenses. In addition, the rapidity of movements can also be overcome by optical means. In comparing this with optical microscopy one could call these methods *time* microscopy. It was found that the motion picture camera possesses the facility of combining optic and time magnification. Therefore the motion picture camera was used in our experiments to record the physical phenomena in the human ear.

There are two principles by which the camera can be used as a time microscope. One is to make exposures at high film speeds. For example, if a motion picture film is run at 64 frames per second and projected at 16 frames per second, the recorded movements appear four times as slow as they were in reality. If, as in high speed cameras, 3000 frames are exposed per second, a playback at conventional 16 frame speed is a time magnification of nearly 200. The high speed camera and the older stroboscope have made it possible to observe the ear during acoustic stimulation.

(Film demonstration of time microscopy.)

Sound has been defined as a train of alternating variations in pressure. For all practical purposes the medium by which the sound wave reaches the human ear is the air. Due to the characteristics of the auditory sense organ, the variations in air pressures must be of a certain strength and at least 20 alterations per second or not more than 15,000 to 20,000 per second. The human ear consists of tissues which are able to vibrate under the influence of sound.

There are two physical prerequisites for the structures of the ear to conduct sound. First, the tissue must possess elasticity; secondly, the tissue must possess mass. In the middle ear the bodies of the ossicles as well as the fluid in the inner ear represent masses. The restoring forces are provided by the fibers of the ligaments, the fibers of the ear drum, round window membranes, annular ligament, and the articulations.

Demonstration: The following terms are explained and demonstrated by motion picture film: mass and elasticity, natural frequency, undamped vibrations, damped vibrations and influence of mass and elasticity on natural frequency.

Natural frequency of the middle ear

Forced vibrations of a rubber membrane

Damped vibrations of a membrane

The ear drum

The concept of phase

Phase difference on model

Demonstration of phase difference between ear drum and round window

The conduction of sound in the normal ear can now be described in physical terminology.

The inner ear is a biologic underwater sound receiver. The normal auditory stimulus is air-borne sound. The ossicular chain, by matching impedances of air and water, avoids the losses which otherwise would occur at the air-water boundary. The lateral part of the conduction system is a receiver of air-borne sound. The medial part, the stapes footplate, is an underwater sound source.

The masses of vibratory bodies in the middle ear cavity are suspended. By being hung in equilibrium on the axis ligament, the effect of the mass is minimized. The sensitivity of the oscillating system is very great.

The natural frequency of the sound conducting system is not placed above the frequency range as modern engineering would most likely choose. Experimentally it was found at 800 d.v. within the most important frequency range.

The conduction of sound through the ossicular chain is a time consuming phenomenon. Compared with the velocity of sound in water or bone, the sound conducted through the ossicular chain shows a lag which can be demonstrated in the motion picture film by the phase difference between ear drum and round window membrane.

PATHOLOGY OF SOUND CONDUCTION

First Case: A Mass Lesion

The air of the middle ear has been replaced by pathologic material. Normally, the tympanic cavity is filled with air. Under pathologic conditions the air is frequently replaced by fluid (otitis serosa) or pus (otitis suppurativa). We are fortunate in being able to show you a rare condition; namely, a soft tumor filling up the middle ear cavity. The case of a 4 year old boy who suffered from glioma is demonstrated. The right ear diseased first; facial nerve and abducens nerve were involved. Tumor tissue was removed from the right ear surgically; the acoustic nerve was cut during the operation (Dr. Theodore Rasmussen). The child was still able to hear with his left ear ten days prior to his death, in spite of the fact that the tumor grew in the left middle ear cavity. The left temporal bone was removed at

autopsy and tested acoustically shortly after in the fresh state. The ossicles were freely moving within the tympanic cavity filled with the soft tumor tissue. Physically, this lesion is characterized by an increase of mass and friction.

The presence of the tumor around the ossicles represents an increase of mass. The sound stimulus has to set into motion not only the normal ossicular chain but additional tissue masses. The condition resembles the physical experiment shown previously. A larger weight is now hanging on the spring than under normal conditions. The inertia is increased. The perception of high tones should suffer most in such a case.

Second Case: A Stiffness Lesion

The second case is that of a female patient who had been seen in the ear, nose and throat clinic for deafness. The diagnosis of nerve type deafness was made. When the patient died of another disease, the temporal bone was removed and tested acoustically. It was found that the stapes did not respond to the acoustic oscillations. From the functional behavior, the diagnosis "stapes fixation" was made. No gross lesion was noticed in the region of the oval window.

The histologic examination showed that the fixation of the stapes was due to otosclerosis. In modern terminology, this condition is considered a "stiffness" lesion.

Third Case: Chronic Inflammation

The third case is a chronic inflammatory condition of the middle ear. The clinical diagnosis was chronic suppurative otitis media with a central perforation in the drum membrane. There were numerous adhesions visible in the middle ear. The patient died of carcinoma. The temporal bones, taken at autopsy, were tested acoustically. The adhesions in the attic and around the short process of the incus can be seen in the film. Similar to case 2, this is also a stiffness lesion. The lesion in case 2 was confined to the stapes footplate, that is, the most medial part of the ossicular chain; while case 3 is a stiffness lesion located in the lateral part of the ossicular chain.

(Demonstration of motion picture, showing the acoustic vibrations in these three diseased ears.)

SUMMARY

The otologist attending patients with impaired hearing loss has to solve two diagnostic problems.

1. What is the extent of the patient's hearing loss?

2. Where is the cause of the hearing impairment located in the ear and what mechanism is the cause?

The solution to the first problem is well advanced due to the development of audiometry. The solution of the second problem is only in its initial phase. Further progress in our knowledge of sound conduction will come if, and when, we learn to understand, analyze and treat the ear as a vibrating system of complicated architecture. The otologist must be familiar with the laws of mechanics and acoustics as extensively as the oculist studies physiologic optics. In this paper the method of testing fresh temporal bones is described as a combined functional and histopathologic study.

The important physical constants of the sound conducting system can be analyzed and related to anatomic structures.

The basic physical components of the sound conducting system are masses and elastic fibers. The diseases of the ear can be studied under these newer concepts as diseases of the mass factor or of the stiffness or friction factor.

Case 1 is predominantly an increase of mass.

Case 2 represents abnormal stiffness in the medial part of the ossicular chain (stapes fixation).

Case 3 is an example of increased stiffness of the lateral part of the ossicular chain.

Low tone loss is associated with an increase of stiffness, while high tone impairment is characterized by an increase of the mass factor. Both low tone and high tone hearing impairment can be produced by conduction lesions. The classic differentiation between middle ear and inner ear hearing impairment as low tone and high tone loss should be abandoned. Our concepts should be replaced by the methods and results of modern physiologic acoustics.

CONSERVATION OF HEARING FUNCTION

JULIUS LEMPERT, M.D.
NEW YORK, N. Y.

ATTEMPTS to conserve hearing function may be classified as follows:

I. The conservation of normal hearing function by avoiding damage to the normally functioning hearing organ.

II. The improvement of hearing function when impairment of such function already exists and the conservation of the improved hearing.

III. The conservation of the already existing functionally impaired hearing by preventing or delaying further impairment of function when improvement is impossible.

I. THE CONSERVATION OF NORMAL HEARING FUNCTION BY AVOIDING DAMAGE TO THE NORMALLY FUNCTIONING HEARING ORGAN

The conservation of normal hearing function should be begun in utero if not sooner. Obstetricians should refrain from inducing labor by administering large doses of quinine which can result in damage to the sensory cells of the organ of Corti in the newborn. The indiscriminate use of pituitrin and thymophosin for the induction of rapid delivery through an ill-prepared perineum or the careless and poorly timed use of the obstetrical forceps may result in intracranial injury with damaged hearing perception. During infancy and childhood the conservation of normal hearing function will depend upon both noncontrollable and more or less controllable factors. The noncontrollable factors which could prevent the conservation of normal hearing function in infancy and childhood are the toxemias accompanying the diseases of childhood, such as mumps, measles, scarlet fever, and meningococcus meningitis, which sometimes irreparably damage the hearing function either partially or totally, monaurally or binaurally. Noncontrollable inner ear infections secondary to otitis media accompanying diseases of childhood could also result in permanently impaired hearing.

The often nonpreventable suppurative middle ear infections with their complications which often accompany the diseases of childhood could prevent the conservation of normal hearing function.

The more or less controllable factors influencing the conservation of hearing function in childhood are mainly:

A. *The avoidance of preventable middle ear infections in the otherwise healthy child.*

The incidence of middle ear infection in childhood can be more or less controlled by:

1. The discriminating removal of chronically infected tonsils.
2. The careful removal surgically of infected or obstructive adenoid tissue without injuring the mucosa of the nasopharynx.
3. The judicious and adequate treatment of suppurative sinusitis when present.
4. Teaching the child how to blow his nose without inflating his eustachian tubes — blowing one's

Lempert Institute of Otology and Lempert Research Foundation, Inc.
Presented at the meeting of the Committee on the Conservation of Hearing, Oct. 9, 1949, Chicago, Ill.

nose by shutting off only one nostril at a time.

5. Avoidance of diving and swimming in non-clean swimming holes or pools—teaching the child that the use of ear plugs in presence of normal tympanic membranes does not prevent middle ear infection.

B. *The immediate and judicial treatment of the middle ear infection when it occurs.*

By early recognition and judicial treatment from beginning to end of an acute middle ear infection, chronicity can usually be avoided and the existing normal hearing function can as a rule be ultimately conserved.

Functional testing of apparently healed middle ears following the exclusive use of antibiotics for the treatment of repeatedly recurring attacks of acute otitis media has led me to the conclusion that from the standpoint of conserving hearing function humanity would be served best if, in addition to the use of antibiotics, early drainage of the middle ear infection through a carefully placed and performed incision of the tympanic membrane by an otologist would also be instituted. Recurrent attacks of otitis media purulenta acuta are much more frequent when treated with antibiotics without drainage than they were when treated with incision and drainage alone. Operating table observations of the pathologic findings which led to cortical perforation of the zygomatic process in the temporal bones of adults and children who were treated with antibiotics only, plus functional testing of hearing in these cases, have led me to believe that if normal hearing function is to be conserved in such ears, humanity would be best served, in the presence of symptoms and signs suggestive of acute suppurative mastoiditis, by employing surgical drainage of the mastoid process through a carefully performed endaural antrotomy plus antibiotic therapy rather than by the use of antibiotic therapy alone.

In adult life the conservation of normal hearing function will also depend upon noncontrollable and controllable factors. Factors beyond our control which could prevent the conservation of normal hearing function during adult life are stapedial footplate ankylosis due to otosclerosis, cochlear nerve degeneration secondary to hydrops of the endolymphatic labyrinth in Ménière's disease, hereditary predisposition to cochlear nerve degeneration, internal ear hemorrhage secondary to injury or hypertension, suppurative labyrinthitis secondary to acute or chronic middle ear infection, chronic adhesive middle ear lesions secondary to chronic middle ear infections, neurosyphilis, pontine angle tumor and acoustic neuroma.

The controllable factors which could prevent the conservation of normal hearing in adult life when not controlled are (1) the ingestion of drugs which could damage the nerve of hearing, such as quinine and streptomycin; (2) being subjected to sound of extremely high intensity for a short period or sound of high intensity for long continuous periods, which may be military, occupational or sportive in character and which could result in damaged cochlear nerve function.

II. THE IMPROVEMENT OF HEARING FUNCTION WHEN IMPAIRMENT OF SUCH FUNCTION ALREADY EXISTS AND THE CONSERVATION OF THE IMPROVED HEARING

In the presence of impaired hearing it is important to determine first whether the hearing loss is of inner ear origin, middle ear origin, or mixed middle and inner ear origin. If the hearing loss is found to be of inner ear origin, hope of improving the hearing must be aban-

doned. There is no known scientifically proved medical or surgical therapy which could improve impaired hearing function secondary to cochlear nerve damage.

In the presence of a middle ear hearing loss due to a nonhealed perforation of the tympanic membrane following a resolved otitis media, the successful permanent closure of the perforation by one means or another can result in improved hearing. In some cases where closure of the perforation is impossible, a properly fitted Pohlman prosthesis may accomplish the same effect.

In the presence of a middle ear hearing loss due to recurrent nonbacterial fluid accumulation of allergic origin within the tympanic cavity, incision of tympanic membrane and removal of exudate with suction will improve the hearing. The determination, if possible, and disposal of the allergic factor responsible for this exudate will prevent its recurrence.

When in the absence of a middle ear infection and in the presence of an intact tympanic membrane the hearing loss which is of middle ear origin is definitely determined to be due to stapedial ankylosis secondary to otosclerosis, it is the duty of the otologist to advise scientifically proved and time-tested measures for improving the hearing. Although I have read reports in the literature and verbally was told of successes in the restoration of hearing function in deafness, whether of middle or inner ear origin, with the use of such remedies as thyroxin, prostigmine, nicotinic acid, histamine, vitamin B complex, Jacobson's solution, eustachian tube inflation, Aix le Bain hydrotherapy plus finger massage and dilatation of the eustachian tube orifice through a rubber catheter inserted into the tube with the aid of a steel bougie, eustachian tube radium application, roentgen therapy of the temporal bone, massage of the tympanic membrane with electric percussor, Urbantschisch phonograph records, lumbar puncture for spinal fluid drainage, tonsillectomy and resection of the nasal septum, and recently with supersonic sound, I personally have never had such experiences. Furthermore, in my 30 years of active otologic practice, I never was able to track down or meet with any such results professed to be obtainable by others. It is, however, today a known, nondisputable and time-proved fact that in middle ear deafness due to stapedial ankylosis secondary to otosclerosis, the Lempert fenestration operation restores and continuously maintains improved physiologic hearing function. This fact is nondisputable, not because you read about it in the literature and not because of verbal hearsay but rather because you saw that happen in your own practice of otology or your own circle of contacts and because the thousands of patients whose improved hearing function can always be scientifically corroborated are acting as living testimony to this fact all over the world. It is therefore the duty of every otologist when consulted by a patient whose hearing function can possibly be improved by the fenestration operation to unhesitatingly recommend this form of relief to him first, last and always since there exists no other treatment today which could improve the hearing function in this type of deafness.

Does the Fenestration Operation When Successful in Improving the Hearing Also Conserve the Improved Hearing?

My experience during the last twelve years with close to 5000 fenestrations has definitely demonstrated the fact that in all those cases where the hearing was improved and the newly created window remained continuously open, the postoperative hearing was conserved without resuming its preoperative course of progressive decline. In May 1948 at the

Royal Society of Medicine of London, I made the following statement,[1] when alluding to the possibility of arresting the progressive nature of the deterioration of hearing function which is characteristic of clinical otosclerosis by the use of the fenestration operation:

> Practical hearing improvements obtained following fenestration in clinical otosclerosis with preoperative evidence of unimpaired cochlear nerve function were as a rule permanently maintained at the practical level when the newly created window remained permanently open. There exists no clinical evidence of secondary cochlear nerve changes in these cases. However, since secondary cochlear nerve changes have only rarely been observed in the non-operated ear of such patients, definite conclusions that the fenestration operation prevented secondary cochlear nerve changes in these cases cannot as yet be drawn.

Further study and analysis of the audiometric charts of my postoperative cases has led to some very interesting and new-to-me observations. As stated and advocated on previous occasions, I always selected for fenestration the ear with the greater audiometric air-conduction hearing loss for pure tone, providing the cochlear nerve function in such ear was still adequate enough to permit restoration of hearing to at least the 30 db. level in the three speech frequencies, which is representative of practical hearing. In the vast majority of instances the air conduction hearing for pure tone in the nonoperated better ear was as a rule somewhere in the neighborhood of the 40 db. level for the three speech frequencies. The interesting observation made was the consistency of the fact that just as long as the air conduction hearing for pure tone in the nonoperated ear did not drop below the 60 db. level in any of the three speech frequencies, no evidence of diminished bone conduction hearing, which could suggest the onset of secondary cochlear nerve changes, was discernible. This observation seems to coincide with the natural course of events as always observed in clinical otosclerosis. The most likely explanation for the fact that in only a small percentage of my postoperative patients did cochlear nerve changes take place in the ear not operated upon is that the time element governing the speed of recession of the air conduction hearing for pure tone in that ear was in most cases insufficient to have permitted its descent to a level lower than 60 db.

In view of the above observations it becomes quite obvious why secondary cochlear nerve changes were never observed in the fenestrated ear when hearing was improved to the practical level and the fenestra remained patent.

It would also seem fair to suggest that since a successfully performed fenestration operation prevents the air conduction hearing for pure tone in the speech frequencies from receding to the 60 db. level, it should result also in the prevention of secondary cochlear nerve changes.

In mixed middle and inner ear deafness secondary to otosclerosis where the loss of hearing is predominantly of middle ear origin, with the bone conduction hearing suggestive of sufficient cochlear nerve function reserve to permit improvement of hearing, though not to the practical level with the fenestration operation, such surgical intervention is advisable in view of the existing evidence that if the fenestra remains patent further degeneration of cochlear nerve function may thus be avoided.

III. THE CONSERVATION OF THE ALREADY EXISTING FUNCTIONALLY IMPAIRED HEARING BY PREVENTING OR DELAYING FURTHER IMPAIRMENT OF FUNCTION WHEN IMPROVEMENT IS IMPOSSIBLE

When the impairment of hearing is due to irreparable pathologic middle ear changes secondary to an existing acute, subacute or chronic temporal bone infection requiring surgical treatment for its

eradication, the otologist must judiciously try to employ various modifications of technic best suited for the individual case in question with an eye towards the conservation of the still existing though already impaired hearing function whenever and wherever possible.

For very many years and prior to the advent of the sulfonamides and antibiotic therapy, the primary concern of the otologist when surgically treating temporal bone infections was to save life, and rightly so. This universally applied philosophy in otologic practice resulted most often in paying too little attention to the conservation of hearing function. Today, with the use of sulfonamides and antibiotics, very little danger to life itself is involved secondary to temporal bone infection, and the otologist should in his treatment of otitic infections give primary attention to the all-important conservation of hearing function.

Just as many men doing otologic surgery are seldom radical enough in the removal of the pathologic processes during their performance of a much-needed mastoido-tympanectomy, so are they often too radical when they unnecessarily do a mastoido-tympanectomy when the complete eradication of the pathologic process requires no more than a mastoido-atticotomy, which can result in the conservation of the existing hearing function as well.

Even in those cases where the pathologic process involves the epitympanum, tympanum and the hypotympanum but not the ossicular chain, which remained intact and capable of functioning, the performance of a limited mastoido-tympanectomy, whereby with careful delicate instrumentation the cholesteatoma, polyps and granulations are removed without disturbing the intact ossicular chain, will result in cure of the infection and in the conservation and sometimes even the improvement of the existing impaired hearing function.

In the presence of a semicircular canal fistula secondary to cholesteatoma within the mastoid antrum and epitympanum, the partially impaired hearing can be conserved if, after removing the foci of infection during the performance of a mastoido-atticotomy, the labyrinthine perilymph space is protected against future infection by covering and sealing the fistula with the tympanomeatal flap.

In pure hereditary nerve deafness of the progressive type, the conservation of hearing will have to await the results of research for the determination and, if possible, the correction of the biologic factors responsible for this form of deafness.

The conservation of the remaining hearing function in patients suffering from nonprogressive partial cochlear nerve function damage of toxic origin following some febrile disease, or when acoustic trauma, or quinine or streptomycin therapy was the cause, will depend upon the hope of and ability in avoiding these etiologic factors in the future.

THE AMPLIFICATION OF SOUND FOR THE DEAFENED WHEN SURGERY TO IMPROVE HEARING FUNCTION WAS EITHER CONTRAINDICATED, REFUSED OR FAILED. ITS RELATION TO THE CONSERVATION OF HEARING

When the diagnosis of chronic progressive middle ear deafness secondary to otosclerosis with good cochlear nerve function reserve is definitely established, it is today the duty of the otologist to acquaint the patient with the fact that he can have his physiologic hearing function improved to the practical serviceable level without risk to life by submitting himself to the fenestration operation. He should also tell him that today his chances of regaining such hearing

function and permanently maintaining it are abundantly in his favor. He should also warn him that there is no other treatment, either medical or surgical, which could improve his hearing function and also that if he does not take advantage of his still existing opportunity to regain his hearing function, the time may not be far off when cochlear nerve changes will manifest themselves and he will no longer be suitable for such surgery and relief therefrom. The otologist should also explain to the patient that if for some reason or other he does not wish to have the fenestration operation, the only alternative that he can avail himself of is the temporary measure of relief offered through the employment of an electric hearing aid. He must be told that the electric hearing aid is not a means of improving his physiologic hearing function but rather a means of amplifying sound for his deafened ear so that he can hear more with it. However, he must be given to understand that his physiologic hearing function will continue to get progressively worse until sooner or later no amount of amplification with the hearing aid may usefully serve his deafened ear.

In chronic adhesive deafness with perforated or absent tympanic membrane, a hearing aid is indicated since fenestration for the present is contraindicated in these cases.

In mixed middle and inner ear deafness where hearing can be considerably improved, but not to the practical level, with the fenestration operation, the possibility of preventing further loss of hearing with a successful fenestration must be weighed against the use of a hearing aid with no such possibility in sight.

In chronic progressive inner ear deafness or in the sudden nonprogressive type of inner ear deafness of either traumatic or toxic origin, where neither medical nor surgical therapy can improve the hearing, both the usefulness of a hearing aid and the advisability of its use are highly questionable. With the use of the aid such patients do not comprehend any more than they do without its use, and whatever they heard comfortably without its use they hear with it much louder, to their great discomfort.

These observations suggest that in nerve deafness certain neural elements have been lost and no amount of sound amplification can bring them back.

How Does the Electric Hearing Aid Affect Our Desire to Conserve the Cochlear Nerve Function Still Existing Prior to Its Use by the Deafened Patient?

Important observations, the significance of which I have recently investigated, were made in patients wearing electric hearing aids.

Patients whose hearing was improved following the fenestration operation but subsequently receded to the preoperative level as a result of fenestra closure sometimes resorted to the use of a hearing aid in preference to having a revision of the fenestra. Since the fitting of a mold for the ear canal of the operated ear was more difficult, they wore the hearing aid in the nonoperated less deafened ear. In some of the ears thus aided cochlear nerve changes manifested themselves within periods varying from two to five years, as evidenced by the recession of both air conduction and bone conduction hearing. With no significant change in the hearing having taken place in the nonaided unsuccessfully fenestrated ear, they were forced to move the hearing aid to the operated ear, which became the better of the two ears for amplified hearing.

Some patients who were operated upon and whose hearing improved but not to the practical level, though the window remained open, were forced to

wear a hearing aid in the better ear, which had not been operated upon, in order to get better binaural hearing. A considerable percentage of these soon showed cochlear nerve changes in the ear thus aided, with hearing by air and bone dropping to a level lower than in the fenestrated ear. They were forced to switch the aid to the fenestrated ear for better results with amplification.

The clinical evidence suggests that when a fenestration is performed for clinical otosclerosis in the presence of already existing advanced cochlear nerve changes in the frequencies 2048 and higher, further spread of cochlear nerve degeneration to the lower frequencies will be avoided as long as the window remains open. When fenestration was not performed and a hearing aid was used the spread of cochlear nerve degeneration to the lower frequencies was often observed.

In fulminating otosclerosis, with evidence of impaired cochlear nerve damage in both the high and low frequencies, the fenestration operation in many instances did not prevent further degeneration of cochlear nerve function. However, when amplification of sound with the hearing aid was resorted to instead of fenestration a much more rapid advance of cochlear nerve degeneration was observed invariably in the thus aided ear as compared with the other nonaided ear.

When in middle ear deafness and in mixed middle and inner ear deafness there was a deterioration of cochlear nerve function following the use of the hearing aid, it was usually found to be in the frequencies which needed the least or no amplification.

In pure progressive inner ear deafness the deterioration of cochlear nerve function in the aided ear was observed to be much more rapid than in the nonaided ear and as a rule was found to be more general and affecting all the frequencies to a greater or lesser degree.

In seeking to explain these clinical findings following the use of the hearing aid, I studied the audiograms taken of such patients before they put on the hearing aid and carefully compared with the audiograms taken after several years of wearing the aid. I also compared the audiograms of the ear in which the hearing aid was not used. The pure tone audiometric findings in the aided ear were observed to follow a definite pattern in hearing depreciation of cochlear nerve origin. This constant pattern of audiometrically observed cochlear nerve loss led me to suspect that the deterioration following the use of the hearing aid was perhaps the result of overstimulation with sound.

Wever and Lawrence[2] summarized for me the effects of overstimulating animals with sound as follows:

I. The ear is injured by overstimulation with sounds.
   a. In animals, the injury is shown both as an impairment of cochlear potentials and as histologic changes in the inner ear.
   b. There are large individual variations in susceptibility to damage. Comparisons may be made either in terms of the absolute sound intensity required to produce damage or in terms of the level of cochlear potentials at which damage appears.
      1. In terms of sound intensity, the variations in normal-appearing ears are of the order of 20 db. (This means that some ears can withstand energies 100 times greater than others.)
      2. In terms of cochlear potentials, the variations are smaller, for normal-appearing ears. These ears begin to suffer damage at intensities that elicit maximum values of cochlear potentials. This means that many of the conditions that determine the the ear's sensitivity also govern its susceptibility to damage. The more sensitive ears are damaged by relatively faint sounds.

3. These conditions are different for abnormal ears. Ears that show signs of an old middle ear infection have poor sensitivity, and require relatively strong sounds to elicit given levels of cochlear potentials.

These ears are damaged before the intensity has been raised high enough to produce a maximum of cochlear potentials.

Ears that have previously been injured by overstimulation are particularly liable to further injury. They are damaged by sounds that normally are altogether harmless. Guinea pig ears that had been exposed to jet engine sounds suffered a further decrease of cochlear potentials of 10 to 12 db. when exposed for 15 minutes to tones only 50 db. above reference level (reference intensity—0.0002 dynes per square centimeter). Normal ears will withstand 80 to 100 db. for hours without damage.

II. The traumatic effects are general.
   a. Single tones cause losses of cochlear responses and of acuity as measured by conditioned response tests for all tones throughout the frequency scale.
      1. For exposure tones below 5000 cycles the losses of cochlear potentials show no particular relation to the frequency of the exposure tones.

         For higher exposure tones the losses of cochlear potentials are greater for the high frequencies, but still are not specific to the exposure tone.
      2. In terms of histologic damage, the results vary according to the frequency of the exposure tone. For low tones there is widespread damage to the apical portion of the cochlea. For high tones the damage is more restricted and is located in the basal portion of the cochlea. Evidently the visible damage is more limited in extent than the functional impairment.
      3. The histologic damage consists of changes in the organ of Corti and particularly in the hair cells. When the damage is great these sensory changes are followed in the course of several days by degeneration and atrophy of the cochlear nerve fibers.

III. An important inference from these observations is that the apparent loudness of a sound is no good indication of its injurious character. A partially damaged ear may be deaf to a particular tone yet may be further damaged by it. Also it should be emphasized that such damage by a tone that is heard only faintly or not at all causes a general deterioration of hearing—a deterioration over the whole frequency scale.

Can the Electric Hearing Aid Overstimulate the Ear with Sound and Damage Cochlear Nerve Function?

An investigation of the highest quality modern electric hearing aids, which was conducted at my suggestion by Wever and Lawrence, brought out the following facts:

1. There exists no possibility in these instruments for amplifying sound for one frequency needing it without simultaneously amplifying for other frequencies which do not need amplification or are in need of less amplification.
2. They do not provide scientifically controlled amplification. Rather they give a highly irregular and highly peaked form of amplification, the result of which is that often there is excessive intensity in the very frequencies needing the least amount of amplification.
3. The claim of some hearing aid companies that the hearing aids which they manufacture and sell can be regulated to amplify only the frequencies needing it, is not substantiated by the tests made with their hearing aids.
4. One of the hearing aids tested when operating at its maximum level produced sound at 135 db. above reference level. Such an intensity of sound is unquestionably dangerous to the ear. In simple nerve deafness where there is an impairment of sound transmission this full intensity will be brought to bear upon the sensory cells of the organ of Corti with injurious effect. In an attempt

to hear, a nerve deafened patient will operate his instrument at or near its full intensity, for since he does not hear the sound, he has no warning of its potential dangers. Sound can reach the inner ear and remain unheard. Sound reaching the inner ear without being heard is more dangerous to the organ of Corti than when heard, since without hearing, whereby the individual may protect himself against it, he cannot measure its intensity and impact.

5. There is no possibility of dampening just one frequency. The controls, if effective at all, only give dampening over a broad range of frequencies.

In the face of the conclusions reached following overstimulation with sound in animals, the above mentioned facts concerning the presently manufactured hearing aids become strongly suggestive that the cochlear nerve function losses observed following the use of the electric hearing aid are in many instances the direct result of overstimulation with sound.

An electric hearing aid can produce acoustic trauma in middle ear deafness because it amplifies even the frequencies which have remained unimpaired or slightly impaired and need less or no amplification to a decibel level which, when used for a long period of time, can injure the hair cells of Corti.

The hearing aid as constructed, manufactured and fitted today can result in overstimulation with sound and cause cochlear nerve function damage.

It is therefore quite obvious, in view of the above facts regarding the presently manufactured hearing aids, that the expression "fitting of hearing aids," which is being employed today, will remain meaningless until hearing aids are built so that the otologist will be in a position to prescribe the necessary degree of intensity to which each pure tone frequency should be adjusted so as to amplify no more than necessary to meet the functional needs of each frequency. Only then will "fitting of hearing aids" carry the same implication for the hard of hearing as fitting of visual aids does for impaired vision.

Humanity will be served best when the hearing aid manufacturers, who are constantly striving to make the hearing aid less and less visible and smaller and smaller, will instead divert their efforts to render the hearing aid more and more useful and less and less perilous, even if the creation of such an aid made it necessary that it be much larger and more conspicuous.

Furthermore, psychologic, social and economic rehabilitation is unwittingly rendered much more difficult for the deafened by the manufacture of small invisible hearing aids. Those deafened who cannot have their physiologic hearing function improved and must resort to amplification of sound cannot reach rehabilitation unless they are psychologically prepared and ready to admit openly and call attention to their infirmity. Once they are mentally ready for such frank admission of their deafened state, they can easily be rehabilitated by the use of amplified sound whether that be obtained through the use of an electric hearing aid, a nonelectrified old-fashioned ear trumpet, a visible cord suspended from the auricle to invite louder conversation, or by simply asking people to speak louder. Rehabilitation of the deafened is hindered by devising ways and means for them to hide their infirmity because they have to live in constant fear of their deafness being discovered, which fear handicaps them mentally, physically and socially much more than their deafness. The more conspicuous the aid, the sooner the rehabilitation.

In addressing an American Hearing Society audience in Rochester, New

York, on how easily one can be rehabilitated with the use of a hearing aid, the charming authoress of "Cotton in My Ears," who was often referred to as the lady who wears a hearing aid which no one is able to detect, prefaced her address by telling how greatly distressed she was before leaving New York for Rochester because she had misplaced and could not find the switch of hair which she wore to hide from view the otherwise visible parts of her hearing aid; she thus unintentionally and unwittingly confirmed my contention that a well-concealed hearing aid is a great deterrent to rehabilitation.

Incidentally, I may add that this lady has just been completely rehabilitated as a result of a successful fenestration operation.

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REGRETS

The officers of the Diversion Club regret that they feel it necessary to call off the meeting of the Club, scheduled for October at French Lick.

There is no sleeper service between Chicago and French Lick, and it is necessary to make part of the trip by bus. The return trip to Chicago is practically an all day journey, and the officers believe that much of the pleasure would be nullified by the tiresome traveling.

We hope to have something to offer next year.

J. WILLIAM WRIGHT, M.D.
Secretary

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Editorial

EMBRYOLOGY OF THE EYE

A motion picture showing the embryology of the human eye will be shown for the first time at the opening of the Scientific Session of the Section on Ophthalmology on Tuesday afternoon, October 10, at 2:20. This motion picture was made at the request of the Council and is based on material made available by the Embryology Department (Baltimore) of the Carnegie Institution of Washington through the courtesy of Dr. George W. Corner, director, and Dr. Vannevar Bush, president of the Institution. It was produced by Sturgis-Grant Productions, Inc., of New York with the aid of Dr. George K. Smelser, professor of anatomy of Columbia University, an experienced teacher of embryology and anatomy of the eye.

The picture, made in color and with sound, has been in preparation for more than a year and demonstrates the chronologic development of the various ocular structures by means of photomicrographs, especially constructed models, and animated drawings.

This picture was made possible only by the combination of fortuitous circumstances. The Carnegie Institution has a remarkable collection of embryologic material that has been well studied. Dr. George Streeter, formerly professor of anatomy at the University of Michigan Medical School, was for several years associated with the Institution in Baltimore and published several studies on the embryology of the eye. Dr. Corner, the present director, very generously agreed to make available to the Academy all specimens, models, charts and drawings for use for teaching purposes. Then the selection of Sturgis-Grant Productions, Inc., as producer brought into the project personnel with experience in medical motion pictures, an adequate staff of artists and the best of equipment.

The success of the project was assured when Dr. Smelser consented to act as technical advisor. He has worked closely with the producer to achieve the utmost in clarity and scientific accuracy of anatomic detail with the result that was desired by the Academy; namely, a

teaching aid in the field of visual education applicable in the basic subjects in medicine. We are indeed deeply grateful to each and every one concerned in the production of this picture.

The cost of this film was borne entirely by the Academy. Reproductions can be obtained through arrangement with the Executive-Secretary. If this venture into the field of instruction in our specialties meets with the reception anticipated, it will give encouragement to the Committee on Motion Pictures to proceed with other subjects. We feel that this project is but another evidence of the interest which has placed the Academy in a position of leadership in medical education.

WILLIAM L. BENEDICT, M.D.

In Memoriam

Francis J. Arnold.....Burlington, Vermont
July 19, 1950

Nora B. Brandenburg.....Winnetka, Illinois
May 31, 1950

Westley Marshall Hunt.....New York, New York
July 28, 1950

Sterling Frederic Mengel.....Pottsville, Pennsylvania
June 2, 1950

Joseph G. Parsons.....Crookston, Minnesota
May 14, 1950

Charles A. Ringle.....La Junta, Colorado
April 22, 1950

Clarence Rubendall.....Omaha, Nebraska
June 6, 1950

Maxwell D. Ryan.....New York, New York
June 11, 1950

Orlando P. Schoofs.....Milwaukee, Wisconsin
June 8, 1950

Gideon McDonald Van Poole.....Honolulu, Hawaii
April 13, 1950

INDUSTRIAL OPHTHALMOLOGY

INTRODUCTORY STATEMENT

The experience, thinking and planning of one of our leading ophthalmologists, who in addition to serving on the Joint Committee is in charge of teaching at one of our great medical institutions, is of utmost importance. The case for industrial ophthalmology in its entirety is presented here by Dr. Edmund B. Spaeth and should be of great value in formulating the policies and technics of ophthalmologists throughout the country.

The use of sulfhydryl other than in chemical eye injuries has been investigated extensively by Dr. Cruthirds, who has broadened the field of its use and intensified the research work in the investigation of how and why this substance acts. He has been especially interested recently in the use of hydrosulphosol in the treatment of corneal scars following ulcers other than chemical burns. For this reason, his paper indicates a step forward in the handling of these lesions so handicapping to the patient. Dr. Cruthirds is presenting his work at the International Congress of Ophthalmology in London and also in Buenos Aires.

HEDWIG S. KUHN, M.D.
Secretary, Joint Committee
Industrial Ophthalmology

INDUSTRIAL OPHTHALMOLOGY

EDMUND B. SPAETH, M.D.
PHILADELPHIA, PA.

THE possibilities of this subject assigned to me for presentation are tremendous. There are so many different phases to it, no one person would dare to speak on the whole field; one is fortunate to be able to discuss a small part of the subject. It probably has three main subdivisions—considering it, for the moment, as a true medical specialty.

The first of these subdivisions might be the organization and administration of job selection and placement, including necessary ophthalmic examinations and the development of technics for such examinations. The second is comprised of the large subdivision of engineering and research for this specialty: problems in lighting and radiation, including all forms of radiant energy, those necessary for a job as well as those dangerous to health; ventilation; the problems of fatigue, space and posture; the chemistry and physics of industry as the various procedures demand human observation and manipulation, and the application to industry of those findings resulting from such research. The third is the protection from injuries, the treatment of injuries when these occur, and the rehabilitation of the injured, that is, their replacement in a gainful occupation. This third portion of the subject can be called the clinical application of the first two.

Industry is interested, basically, in the manufacture of some product and the disposal of this for profit. This applies in all branches of industry regardless of whether it be connected with a private firm, as an automobile factory, with the machine shop of a government navy yard, with heavy transportation, or with road building. Management is not interested in any item which will raise the cost of manufacturing of its product and/or fulfilling its contract unless that additional expenditure will result, in the long run, in increased production or in lowered costs. This is only natural and perhaps that policy must continue to be necessary. Employer, employee, and ophthalmologist are all essential for this program. The employer must be convinced that time spent in eye examinations, the cost of medical and surgical supplies, protective appliances, research laboratories, and medical staff salaries—to quote the most obvious fac-

tors—are justified. The employees must be protected against themselves, a project of education for management and for medical staffs. This is basic, for it is a well-known fact that 40 per cent of all individuals, thus all potential industrial workmen, have visual deficiencies even before employment. Equally significant is the statement that 90 per cent of all industrial accidents could be prevented by proper equipment, using those words in a very broad sense. Last, in this matter of responsibility, is the ophthalmologist. Too frequently his education, hence his interest, is only in the treatment of accidents rather than in the prevention of them. Education and training are necessary before the ophthalmologist can cope with the special problems of any specific form of employment. Very simple problems may be considered here as illustrations. In some occupations color vision is not essential, in others excellent color discrimination is required, and in a third good stereopsis is the most important visual essential. The only common factor each of these three present is the necessity for ophthalmic screening.

The problems of the industrial ophthalmologist are the following: the determination of procedures necessary before a person is employed, procedures necessary when or after being employed, and the prevention and treatment of injuries.

The first of these includes, as essentials, aid in the establishment of a medical staff; the institution of a system or method of examination and screening for both applicants and employees; provisions for rechecks at stated intervals; the development of suitable systems for recording an individual's history and the findings from the tests and examinations, for these data are an aid in the proper placement of all new and present employees; and assistance in placement, for visual qualities and abilities are not possessed to a maximum degree by all employees even with the best of correcting devices. The ophthalmologist must indicate to the placement authority those jobs suitable to the best obtainable visual and oculomotor efficiency of each employee, keeping in mind efficiency as well as the risks of injury to himself and to others. This implies a huge task of job classification.

The ocular examinations necessary and applicable when the individual is employed are somewhat different. Some of these, which are to be emphasized, follow. The study of occupational lenses is most important, for the age of the skilled worker is steadily and certainly increasing. The demands made upon vision by modern industry vary greatly with the different types of employment, so the industrial ophthalmologist must consider securing optimum vision at varying distances depending upon the type of employment. One example of this is quoted as an illustration. Donald P. Bixler,[1] in establishing an eye-corrective as well as an eye-protective program in 1947 at the direction of the Under Secretary of the Navy, stated:

> During this survey one important fact was noted; namely, that although many presbyopes were properly corrected, when they put on certain protective devices, notably welding hoods and burners' goggles, they were unable to utilize their bifocal segments in doing their near work. Some had separate near point additions incorporated in their hoods and goggles, but these were in the distinct minority. The usual sources of optical supply did not have any devices to overcome this defect. The question thus arose as to how a worker doing a near point task could do it efficiently and well if he could not see properly.
>
> We found that this problem was solved satisfactorily by having a large, flat, uncut, spherical lens cut rectangularly to such a size as to fit in the window of the welding hood. This then took the place of the unusable segment addition in the goggle lens. It was found to be an easy matter to have a lens cut of such size and power as to closely approximate the interpupillary distance and the effective

power which would exist at the new corneal hood lens distance. This same method was used for burners and others using cup goggles for near work.

The study of adequate illumination, as earlier mentioned, as well as the control of unusual types and degrees of the various forms of radiant and atomic energy, is equally important. The study of the relation of vision to general health, and the opposite, includes the control of ventilation and the control of noxious fumes and atomized particles, of posture at work, of nutrition, and frequently of domestic and social relations. Suitable arrangements must be made for the re-examination of those found to have subnormal visual abilities, of those with high accident rates or with low production outputs, and for the correction of such defects and findings. This frequently includes the necessity for furnishing administration with recommendations for such situations. The institution of protective programs and campaigns, of various forms of protective devices, and of methods for their continued application is rather similar to, in fact a part of, the program just mentioned.

Procedures which fall within the so-called clinical side of industrial ophthalmology are not especially complicated. First aid stations must be outlined and established, easily accessible with the necessary equipment and medical supplies for the examination and treatment of eye injuries. It is probable that a nurse should be in attendance at all times; one who has been especially trained and instructed is most valuable. Her activities should be under the direction of the ophthalmologist at all times. If the size of the plant is too small for a nurse to be in attendance, then some employee should be given training in first aid.

Arrangements must be made for the transportation of injured patients to a hospital or infirmary where first aid forms of therapy are terminated and other necessary medical, surgical, or investigative procedures are carried out. Recovery of these patients should be completed by placing them in proper jobs, after necessary training and the consideration of their added visual handicap. The industrial ophthalmologist should be capable of giving these patients advice in the medicolegal field as to the computation of the degree of visual disability resulting from the injury, and he should have an expert knowledge of the compensation laws of the several states.

The benefits which appear from professional eye care for workers with lowered visual performances are remarkable. There can be no doubt any longer about that. Drs. W. Gregory Morgan and N. Frank Stump[7] recently completed a detailed survey of those benefits. Two basic facts were revealed. First, there is a direct relationship between visual performance and average hourly piece-work earnings; and second, there is a definite advantage to the company when visual performance standards for various jobs are met. Some of the conclusions of these two authors follow herewith, verbatim.

> Very definitely, proof is now available that a new employee is, at least, visually qualified when he goes to his new job.
>
> In experienced employees, those who meet the minimal visual qualifications average more money per hour (from four to nine cents) with less make-up pay than those who do not meet the standards.
>
> Training time is less when there is increased efficiency in visual performance, savings in learning time ranging from 12 to 35 per cent.
>
> Re-surveying of visual performance can be done any time when the need is apparent.
>
> The visual performance tests form a very important part of our physical examinations. Many more applicants for employment are required to receive professional attention because of visual inefficiencies than any other single factor. All applicants who have ineffi-

cient visual performance are urged to seek professional eye care and thereby many are salvaged and, subsequently, brought up to the minimal requirements.

Present employees who did not meet minimal requirements and who, on our advice, sought professional eye care, later as a result made more money for themselves as well as for the company.

In transferring an employee from one section to another, a check is made to see if he is visually qualified for the prospective job. A "vacancy-posting" set-up is in operation. By this procedure the employee, generally, initiates the request for a transfer. Then a check is made to determine whether he passes the medical examination and the visual performance tests.

In our program every effort is made to carry out the basic philosophy, "Eyes for the Job."

Sufficient controls were set up by these investigators to indicate that the improvements obtained, and quoted briefly above, were largely due to an increase in visual performance. There are no limits to management's co-operation nor to the lengths to which ownership and management will go when presented with such evidence.

As a second portion of this paper—the first, it is obvious, having to do with administration and basic principles—the author wishes to discuss some important aspects of the clinical application of industrial ophthalmology. Those selected have been chosen because of their certain constant importance. They are the necessity for the posting of "Standing Orders" in plants, in infirmaries, and in dispensaries; the better treatment of chemical burns of the eye and lids; some factors in intraocular metallic magnetic foreign bodies; and the disposition of the uniocular aphakic.

Many plants have inconstant personnel at their first aid stations. Some may have full-time physicians, while very small plants may have to depend upon the office secretary for first aid treatment. Nevertheless, including both extremes, standing orders are of outstanding importance. Thomas W. Nale's outline[8] of the orders of the Union Carbide and Carbon Corporation of New York is a classic example of this. He will be quoted extensively and, in part, verbatim. His orders include five subdivisions. They are: (A) Foreign Bodies Entering the Eye; (B) Traumatic Injuries — Lacerations or Puncture Wounds; (C) Flash Burns or Thermic Burns of the Eye; (D) Persons with Hot Pieces of Metal or Sparks in the Eye; and (E) Chemical or Irritating Fumes, Exposures and Contacts.

In the qualification of these orders great attention is paid to adequate examination. This sounds almost absurd when addressing physicians who are in the greatest number also clinicians. Nevertheless, a lack of adequate initial examination is the outstanding fault in treatment of these cases. Such factors are emphasized as everting the upper lid, staining all cases with fluorescein, the use of adequate illumination, the examination of an eye for the expulsion of fluids from the eyeball itself (a sign of perforation of the eyeball), the observation of the pupil in ocular contusions, the questioning of the patient as to the vision still present in an apparently damaged eye, and the recognition of the fact that certain chemicals can and do cause delayed reactions instead of immediate effects, and that one must be on the lookout for these. Chemicals in liquid or in gaseous form are equally dangerous if the patient suffers exposure to them.

A survey of blindness from industrial accidents shows that some form of a chemical trauma to the globe is still the single highest cause for industrial blindness or severe impairment of vision. There is no doubt that the prevention of these injuries is improving. As long as they appear, however, adequate and proper treatment is of outstanding im-

portance. The requirements for such treatment are threefold: (1) the dilution and through this the removal of the chemical; (2) the chemical or physical neutralization of the offending substance, but even more important, a neutralization of the effects of the substance upon the tissues involved, and (3) the protection of the adjacent normal tissues and the recovering tissues, as well as the stimulation of the regrowth of those tissues involved. To carry out these essentials in treatment it is necessary to know the offending substance, the approximate degree of exposure, and the length of time which has intervened between exposure and the first treatment. It is wise to remember that many gaseous substances penetrate the cornea rapidly, especially the ammonium radical, and that repeated anterior chamber paracenteses must be done to limit the anterior chamber damage from the intraocular aqueous solutions of those chemicals.

These cases, when initially injured, are not bacterially contaminated, but that can and frequently does follow rapidly because of the rapidity of bacterial growth upon necrotic tissues. Water is the best diluent in almost all situations, and usually an adequate amount of this is easily obtained and accessible. The dirt almost certainly present and connected with the original injury and the subsequent immediate treatment are usually the causes for these infections. One should remember this without fail in considering the first aid of ocular injuries.

Dr. Hedwig Kuhn[4] calls attention to the obsolete practice of treating acids with bases, and the opposite, that is, the chemical neutralization of substances rather than the neutralization of the effects of such substances upon the tissues. A solution of neutral ammonium tartrate is the only chemical substance still considered as satisfactory and adequate for the first of these demands. Actually it continues to be a most valuable therapeutic agent. The second, though this in effect bridges that question of chemical neutralization and the neutralization of the effects of chemical substances, is the surgical resection of necrotic tissues and the immediate use of mucous membrane grafts as replacements for these necrosed tissues as well as for the protection of the sensitive contiguous tissues which would certainly become infiltrated with scar tissue, hence lose transparency and become vascularized. In so far as treatment of the effects of chemicals upon tissues is concerned, the use of the sulfhydryl treatment as outlined by Cruthirds, [2,3] and described recently and in further detail by Kuhn,[4] is almost ideal. This sulfur preparation, used as hydrosulphosol, because of its chemical composition stimulates promptly the healing of tissue injury produced by the accidental exposure of tissues to the damaging action of heat and of chemicals. As Kuhn said:

> In a search for a local wound function for the sulfhydryl radical, Localio and his co-workers[6] point out that . . . there exist in a protein molecule certain bonds and groups in the side chain without whose integrity enzyme activity does not exist. Of these groups the electronegative sulfhydryl (SH) group has been shown to be of much importance. They have shown that many of the reactions of the metabolic cycle are catalyzed by SH containing enzymes.

These SH enzymes, furthermore, exert (as Localio and his co-workers stated) their most important function during the early stages of healing. This is of great importance in burns of the cornea and of the lower cul-de-sac.

Three of Dr. Kuhn's conclusions follow herewith:

> Results observed [in more than 300 cases] include ability to control pain at all times, produce faster healing and effect recovery

with less impairment of vision as a permanent disability and fewer occasions when conjunctival flaps were needed or contractures resulted.

The use of this material was based on its apparent value as a specific treatment for eye burns because of its chemical composition and an indicated ability to stimulate healing functions related to biochemical aspects of the structure of this particular organ.

The simplicity of technic adds to its attractiveness.

The use of this substance for injuries due to anhydrids and to detergents has shown similar results, though this is still under investigation by the resident staff of the Graduate Hospital of the University of Pennsylvania, as well as at other research centers.

In considering the therapy for many aspects of the intraocular metallic foreign bodies, one too frequently underestimates the value of a history of the injury. This should include not only a review of the anatomic conditions present at the time of the first examination but also the course and pathway of the moving metallic fragments and a careful examination of the possible metals of which the intraocular foreign body could consist. A foreign body which has resulted from the removal of a bushing from a bearing with a cold chisel and hammer, for example, could be either high carbon steel, brass, or low carbon steel, and each of these three, it is very evident, has a different response to a magnet. The prognosis for intraocular magnetic foreign bodies is so much better than for nonmagnetic particles that differentiation is desirable. The density of radio-opaqueness as seen upon a roentgenogram is not sufficiently satisfactory for determining magnetic qualities. The treatment of the two possibilities is so very different that one must learn as much as possible about each case.

Urgency in the transportation and surgical treatment of these cases is outstanding. The antibiotics now available and almost selective as to the type of organism present, combined with this factor of early surgery, are largely responsible for the wonderful visual results being obtained after the successful removal of these foreign bodies.

Posterior route magnet extractions are to be limited to those cases in which the foreign body can be removed through a posteriorly placed wound of entrance, to cases in which the foreign body is too large or of such a shape as to make it quite inadvisable to carry it through the suspensory ligament, and cases in which there seems to be good evidence that the foreign body is entangled in the retina, in the choroid, or in both. These cases need accurate localization, for the foreign body should be removed through a posterior sclerotomy accurately overlying the foreign body. The importance of accurate localization, by some form of roentgen examination, when the foreign body is otherwise only behind the lens-suspensory ligament diaphragm is much overestimated. All one needs to know is that the foreign body is magnetic and that it is free within the vitreous. These cases ought to be operated upon with the giant magnet. By means of this magnet the foreign body is to be passed through the suspensory ligament into the angle of the anterior chamber. At this point the hand magnet should be substituted for the giant magnet, and the foreign body removed after a keratome incision into the anterior chamber angle at the root of the iris. The hand magnet is essentially a contact instrument and is used with the assumption that no more than the thickness of the cornea or the sclera lies between the tip of the magnet and the foreign body. The giant magnet is used for entirely different reasons, in quite a different manner, and under principles which are quite different. One must pay attention in the use of this instrument to the magnetic field and to those laws

which are distinctly applicable to the magnet as well as to the foreign body. Success with the use of the giant magnet will be certain only under such circumstances. That practice of introducing a long slender curved tip on a hand magnet into the vitreous is fruitless and very poor surgery as well as bad physics.

It seems tragic, but apparently it is still necessary to repeat that statement—originating from many sources—which has appeared so frequently in the literature, that an original scleral wound of entrance as well as the wound of a posterior sclerotomy should be treated with diathermy or with actual cautery to prevent the development of a later retinal separation due to that sclerotomy incision into the retina or to the retinal laceration by the entrance of a foreign body into the vitreous chamber.

The lot of the uniocular aphakic is at the present time accompanied by doubt and by considerable difficulty. Two years ago the Pennsylvania Superior Court ruled that an eye shall be considered lost, for compensation purposes, following the extraction of a traumatic cataract. Because of this unwise decision insurance companies will refuse to pay for the treatment of an injured eye in which a traumatic cataract may develop because of the injury, knowing that regardless of the operative result obtained they will have to pay for the complete loss of that eye. The effects of this ruling are all tragic, and some are so absurd as to be exasperating. Two examples will illustrate. The first is that of a one-eyed man, now with 6/6 vision with his aphakic lens, receiving compensation for the total loss of this remaining eye, who is now gainfully employed as a carpenter. The second is that of a coal miner, receiving payment for the total loss of both eyes, who has 1/60 and 6/6 vision in the two eyes, respectively, and who faces the probability of being paid twice for the loss of the same eye because of a second injury to the right eye. Other cases illustrative of the stupidity of this court decision will appear.

At the present time a case is being carried through compensation referee hearings, through the Court of Common Pleas, and to the Superior Court to have this decision reversed, if possible. The degree of compensation payments to be awarded should depend upon the best central visual acuity obtained with the best optical aid possible, upon the size of the remaining field of vision, and upon the presence or absence of an intractable diplopia. This will permit the evaluation of corneal, retinal, and oculomotor defects as they affect visual capacity.

Binocular single vision is a common finding after the extraction of cataract from one of the eyes. The use of a contact lens has proved this without any doubt. Even that is not necessary, however. Hugh A. and Hedwig S. Kuhn[5] analyzed 82 histories of patients who had been operated upon by them for monocular traumatic cataracts and who were fitted with their aphakia correction for constant wear. Naturally, the largest number of these cases needed training in learning to use their monocular aphakia lens, and such training was supplied them until they were comfortable or it was found that they could not achieve comfort. Sixty-eight, or 83 per cent, of the patients had no great difficulty in finally being able to wear their full aphakia correction. Eleven patients, or 13 per cent, had a moderate amount of trouble but were not sufficiently uncomfortable to require a change or to dispense with the glasses. Only 3 cases were totally unable to wear their full correction, and of these one was made comfortable with a contact lens.

The reasons for insisting upon surgery in these cases, as presented by the Kuhns, is concurred in without question by this author. These are:

1. Occupational demands requiring nearly normal vision in both eyes.
2. The inherent right of an individual to be given the best possible vision.
3. The advantage to the surgeon and the patient that is gained by an operation of choice when the patient's physical condition is good.
4. The actual relative infrequency of any great postoperative discomfort.

There is little doubt, considering the above, as to the wisdom of the Joint Committee on Industrial Ophthalmology in preparing a resolution for approval by the American Academy of Ophthalmology and Otolaryngology as to this questionable court decision mentioned above. It is hoped that this organization will approve such a resolution.

CONCLUSIONS

An attempt has been made in the presentation of this paper to emphasize the necessity for better ophthalmology in industry and to show the opportunities present for this. In addition, various important clinical applications have been discussed in that they seemed to be most significant in a presentation which must be as brief and as limited as this.

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USE OF SULFHYDRYL IN THE TREATMENT OF CORNEAL SCARS FOLLOWING CHEMICAL EYE BURNS AND ULCERS

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CLEARING up of heavy corneal scars with restoration of normal or near-normal vision has been accomplished by a combination of topical and oral use of sulfhydryl. Search of the literature indicates that this is the first time such a result has been achieved.

So unexpected were our results that other ophthalmologists using this same sulfhydryl therapy were queried. Therefore, we are able at this time to add reports of comparable results obtained by at least three other clinicians, all located at widely separated points.

This use of sulfhydryl, as contained in products which are identified by the trademark "Hydrosulphosol,"* was prompted by its successful application in hundreds of cases of major eye burns. Two doctors alone have each treated more than one thousand such injuries. The clinical results observed in the treatment of major chemical and/or thermal eye burns have been covered in a series of nine published reports. However, we

*Hydrosulphosol is the registered trademark of the E. C. Lientz & Co., Inc., Fillmore, Calif., for their brand of sulfhydryl.

wish again to emphasize that, when this medication is used to treat new eye burns, the development of scars, adhesions and other ocular complications can be almost entirely avoided.

The importance of having available for immediate use an effective therapeutic agent for chemical eye burns is being emphasized more and more. With the introduction of a wide range of new chemicals into daily use in the home, on the farm and in industry, the hazard of sustaining chemical eye burns is greater than ever before. Every means possible should be taken to alert the public to the dangers that exist should any one of a number of such chemicals get into the eyes. Every minute of delay in obtaining proper treatment from an ophthalmologist may lessen the chances of saving vision and, in some cases, even the eye itself.

Having demonstrated the outstanding efficacy of sulfhydryl for eye burns, we turned our attention to the possibility of employing this same healing agent in the treatment of heavy corneal scars. Scars can result from inadequate treatment of eye burns with other methods and many are left after treatment of corneal ulcers.

In the treatment of old, thick corneal scars, urgent need is presented for a supportive systemic therapy in addition to employment of topical applications. It may seem remarkable that hydrosulphosol can be used both topically and orally, but ophthalmologists fully acquainted with this medication do recognize the potentialities. At any rate, the clinical responses elicited by the local application of hydrosulphosol suggest that the theoretic biochemical considerations involved provide a plausible background for its oral use. Certainly these new clinical findings cannot be brushed aside lightly.

The discovery that this chemical agent will clear up heavy corneal scars and restore normal or near-normal vision to patients heretofore regarded as inescapably doomed to loss of eyesight holds great promise in the field of ophthalmology. Because of its importance, we will first present the clinical evidence and follow with an explanation of the theoretic biochemical aspects involved.

Case 1 presents data on a result achieved entirely by local application of ophthalmic hydrosulphosol oil. We are indebted to Dr. Hedwig S. Kuhn[7] not only for this case history but for the development of this particular ophthalmic medication that has proved so spectacularly useful in the treatment of major chemical eye burns. In this connection, it can be noted and emphasized again and again that when this ophthalmic hydrosulphosol oil treatment can be applied promptly to the most severe cases of chemical eye burns, the distressing sequelae of scars, adhesions and serious ocular complications can be prevented. Until something more effective is developed we have no hesitancy in urging the use of this medication and technic as developed by Dr. Kuhn in the treatment of every chemical eye burn.

We are indebted to Dr. W. H. Belknap[3] for the histories here presented as cases 2 and 3. Dr. Belknap applied this sulfhydryl product successfully in well over a hundred eye burns before he used it to treat corneal scars. Because of the depth of the scars and the percentage of vision recovered, Dr. Belknap's cases possibly are among the most spectacular presented.

Two other cases, 4 and 5, involving healing of corneal scars are taken from our own series. These patients had consulted a number of other clinicians before coming to us, and one of these patients was facing loss of the left eye.

Case 6 is presented through the courtesy of Dr. Joel P. Smith[11] of Atlanta, Georgia. The implication here would be

that hydrosulphosol is capable of stimulating specific sulfhydryl groups of the cornea which are associated with pain sensation and lacrimation.

All of these patients had been under observation for varying lengths of time, from several weeks to several years. Blood and other laboratory tests were quite complete and no unusual complications, beyond what would ordinarily be expected, were noted except as will be referred to briefly in the following case histories.

CASE HISTORIES

Case 1

Male, age 51. Scar extending over the pupillary area due to severe lye burn of right eye. Patient treated elsewhere for 18 days without improvement before entering Kuhn Clinic. Eye extremely painful; deep staining of cornea and conjunctival surfaces. Iris not visible.

Treatment: Ophthalmic hydrosulphosol oil using pressure bandage technic. Supportive therapy—riboflavin and vitamin E by mouth.

Result: After 19 days (24 hours after entering Kuhn Clinic) eye was left open without medication. Cornea cleared by end of 40 days, and scar reduced to an area below the pupil. Patient, who flies his own plane, can fly again and has a vision of 20/30 unless he looks straight down.

Comment: When treatment was started damage to cornea was such that it was not expected that the eye itself could be saved.

Case 2

Female, age in the sixties. Scarring of the cornea following multiple ulcers. The scars were heavy and had been present for about 38 years. Vision was less than 20/100.

Treatment: Hydrosulphosol, 10 drops orally in gelatin capsule taken after each meal. Dilute hydrosulphosol solution in drops in eye daily.

Result: General condition of patient showed early improvement but no change in scar was noted until after approximately six months, when scar began to thin. Following this, scar almost entirely disappeared. Vision, with correction, has returned to 20/30 with a very pronounced improvement in the appearance of the eye and especially the cornea.

Comment: This was an unexpected result as the scar, which developed over a period of 38 years, had grown heavy with gradual loss of vision despite any treatment previously applied. Administration of hydrosulphosol for oral use is being continued to see if the scar will disappear entirely.

Case 3

Female, age in sixties. This case is regarded as being slightly more spectacular than case 2. A thick, ugly scar was present in the central pupillary area following a dendritic ulcer. Vision was less than 20/80.

Treatment: Hydrosulphosol for oral use, 10 drops in a gelatin capsule taken after each meal, started about four months after the eye was quiet.

Result: After five months from start of treatment the scar had almost disappeared. Vision 20/20 with correction.

Comment: Treatment is being continued with hope of complete recovery.

Case 4

Female, age 19. Iritis and iridocyclitis. Patient had been under treatment elsewhere for 45 days. Complained of burning and itching in left eye with no vision in that eye. Right eye showing signs of involvement. Enucleation of left eye had been recommended. When first seen by us entire cornea of left eye was covered by ulcer.

Treatment: Badly infected tonsils removed. Ophthalmic hydrosulphosol gel used once a day, also hydrosulphosol solution, 1:40 in distilled water, 2 drops every two hours in left eye. Hydrosulphosol for oral use, 5 drops in a gelatin capsule taken after each meal.

Results: Scar on cornea of left eye reduced to one-third original area by end of six weeks with vision continuing to improve. Right eye completely cleared up few days after start of hydrosulphosol therapy.

Comment: Response in this case was prompt and clearing up of scar and return of normal vision in left eye is expected with continuance of treatment.

Case 5

Male, age 31. Loss of vision in left eye due to vernal catarrh. Was under treatment elsewhere for seven months before being seen by us. When first seen vision of left eye was blurred hand movement at 6 feet. Right eye had been removed 17 years before as result of injury due to small lead shot in eye.

Treatment: Hydrosulphosol for oral use, 5 drops in a gelatin capsule taken after each meal; ophthalmic hydrosulphosol gel once a day, and 2 drops of 1:40 hydrosulphosol solu-

tion diluted in distilled water every two hours in left eye.

Results: Cornea clearing, vision gradually returning with improvement in the appearance of the eye and especially the cornea.

Comment: This patient is a veteran and had been hospitalized in the east before arriving in Arizona. Plans had been discussed with recommendation of corneal transplant. It is too early to report final outcome of this case, but treatment is being continued with the prospect of eventual clearing up of condition and return of good vision without need for surgical intervention.

Case 6

Female, age in the sixties. Bilateral corneal dystrophy. Patient has been blind over three years, with extreme lacrimation since onset of condition.

Treatment: Ophthalmic hydrosulphosol gel in both eyes once a day.

Results: Under treatment about six months. Extreme lacrimation and photophobia have practically stopped. No response of the dystrophy noted as yet.

Comment: Treatment being continued.

Supplementing the foregoing case histories it might be noted that we reported in 1946 successful healing of a severe case of corneal ulcers.[4] Hydrosulphosol solution, 1:40 dilution in distilled water, was used as drops in the left eye every two hours and compresses saturated in a 1:20 dilution of hydrosulphosol solution were applied twice a day. Pain was relieved within the first 24 hours and ulcers were healed by the end of two weeks. However, a large central corneal opacity which extended over the central and lower paracentral cornea was left. Subsequent treatment with drops in the eye and compresses saturated with 1:20 hydrosulphosol solution was followed with clearing of the corneal scar until the scar was practically invisible to anyone standing two feet away. The hydrosulphosol product prepared for oral administration and the ophthalmic hydrosulphosol oil, used in the later cases here reported, were not available in 1946. Results suggest that the oral preparation is indicated, along with topical applications of the ophthalmic product, in older cases at least.

SUMMARY

In a brief review of the sulfhydryl therapy as used in the treatment of major eye burns and corneal scars, the use of the oral preparation with local application of ophthalmic hydrosulphosol products is indicated as providing a valuable combination treatment in all corneal scars. Also, it is suggested that in severe eye burn cases where the general resistance and condition of the patient is low or healing is slow, hydrosulphosol for oral use could be a supporting treatment to the application of ophthalmic hydrosulphosol oil, which is the local treatment of choice.

A considerable amount of information has been published which suggests that the use of a sulfhydryl preparation such as hydrosulphosol is capable of influencing the activity of SH groups in ocular tissue. Recent studies reported by Sullivan[12] indicate rather clearly that hydrosulphosol acts in vitro as a true organic sulfhydryl would be expected to act, even though we know that, as contained in hydrosulphosol, the sulfhydryl does not have any organic linkage. In animal experimentations the evidence is that hydrosulphosol acts in vivo exactly as it acts in vitro so far as the dissociation of alloxan is concerned. This suggests the possibility that the chemical activity of hydrosulphosol causes it to combine with some other compound in the body and thus endows it with a therapeutic activity that it did not previously possess.

Sullivan reports that hydrosulphosol acts similar to BAL in protecting rats against the fatal convulsive effects of alloxan. He also notes that "rats receiving 0.11 cc. of hydrosulphosol did not display any toxic effect of the drug and remained active indefinitely," whereas a

group receiving 0.03 cc. of a 10 per cent dimercaptopropanol were "not as active as those receiving an equivalent amount of sulfur as hydrosulphosol but later recovered."

In the many studies reported on the testing of BAL as a treatment in arsenic and mercury poisoning, it was pointed out[9] that "arsenic and mercury combine with and block the function of the physiologically essential groupings in the cell, specifically of cellular SH (sulfhydryl) groups. BAL competes with these cell constituents for the heavy metals and in the case of arsenic forms a stable combination, thus permitting the removal of arsenic from the tissues. The excretion of the BAL-arsenic compounds is rapid, so that the body is quickly freed from the toxic agent." Also, it is pointed out that following the use of BAL, which acts as a reducing agent and contains sulfhydryl, improvement occurred with astonishing rapidity.[6]

In the same editorial another important point is emphasized.

> The significant results that have emerged from the studies on the action of BAL are the remarkable curative property in the treatment of poisons caused by certain heavy metals, such as arsenic, mercury, gold and antimony, and, no less important, the advance in the theory that a biochemical lesion in a tissue formed by partial blocking of an enzyme can cause pathologic damage. It is likewise significant that this pathologic change can be proved to be reversible.

In the alloxan experiment, the fatal convulsive effect produced by injection of this diabetogenic in rats is due to selective necrosis of the beta cells of the islets of Langerhans of the pancreas. Injection of BAL 10 minutes before injection of alloxan protects the animals. In a similar experiment it has been found that injection of hydrosulphosol solution in rats 10 minutes before injection of alloxan also protects the animals.

Since it has been reported[1] that a significant decrease in the lenticular glutathione has been observed due to the effect of heat on the eye, there is basis for the assumption that a state of sulfhydryl inadequacy can develop in cases of major eye burns. There is also the well-founded theory that, when healing of an injury is delayed, a stimulation of activity of the body sulfhydryl may be produced by the administration of dl-methionine.[8] In these tests the methionine was selected because it contains sulfhydryl.

Hydrosulphosol, which contains a concentration of sulfhydryl ion approximately fifty times greater than is present in human blood,[10] has acted spectacularly to heal both new and old burns. The agents causing these burns appear to cover virtually every such hazard known. Included in old, nonhealing lesions that have been healed under the influence of hydrosulphosol therapy when all other measures had failed, have been a large number of x-ray burns.[11]

Highlighting the important place occupied by sulfhydryl in the human economy is the following statement by Barondes and Levine[2] in a report on "Atomic Radiation Effects":

> Sulfhydryl compounds are essential tissue constituents and of great import in the biologic phenomenon of body detoxification, oxidation-reduction, cellular respiration and oxygen capacity. (Inorganic sulfur compounds lack detoxifying properties and would serve no useful purpose here.) The sulfur-containing bodies having sulfhydryl form as cystine and methionine, are the sources of glutathione which is most necessary for cellular respiration and life itself. Quite apart from the ordinary duties of the body, organic sulfur is called upon when emergencies arise to combine with toxic substances which are ingested or that arise from some perversion of the tissues, e.g., that from prolonged intensive radiation. Sulfhydryl compounds should find a prominent place among the prophylaxis and therapeusis here. The state of sulfhydryl inadequacy (hyposulfhydrosis), the result of increased demand for additional sulfur in the detoxifying processes, constitutes a pathologic entity and is evident in the light of knowledge at hand.

CONCLUSIONS

Disappearance of heavy corneal scars and restoration of normal or near-normal vision has resulted following application of sulfhydryl therapy in a series of 5 cases. When the conditions encountered and results achieved have been fully studied, this treatment appears to open a new, effective line of attack on some problems that have heretofore resisted all therapy.

A sixth case, involving corneal dystrophy, indicates possible favorable reaction of hydrosulphosol therapy in stimulating specific sulfhydryl groups of the cornea associated with pain sensation and lacrimation.

Application of a sulfhydryl compound to the treatment of conditions generally classified as presenting "irreversible tissue damage" was prompted by results observed when using this therapy successfully on major chemical eye burns. Until this treatment was applied to eye burns the most severe cases would previously result in scars, adhesions and damage of a permanent nature. With this sulfhydryl therapy applied locally even the most severe chemical eye burns now heal quite promptly. In the study reported here a new sulfhydryl product prepared for oral use has been combined successfully with local applications in the treatment of old, thick corneal scars that have proved resistant to all other forms of medication.

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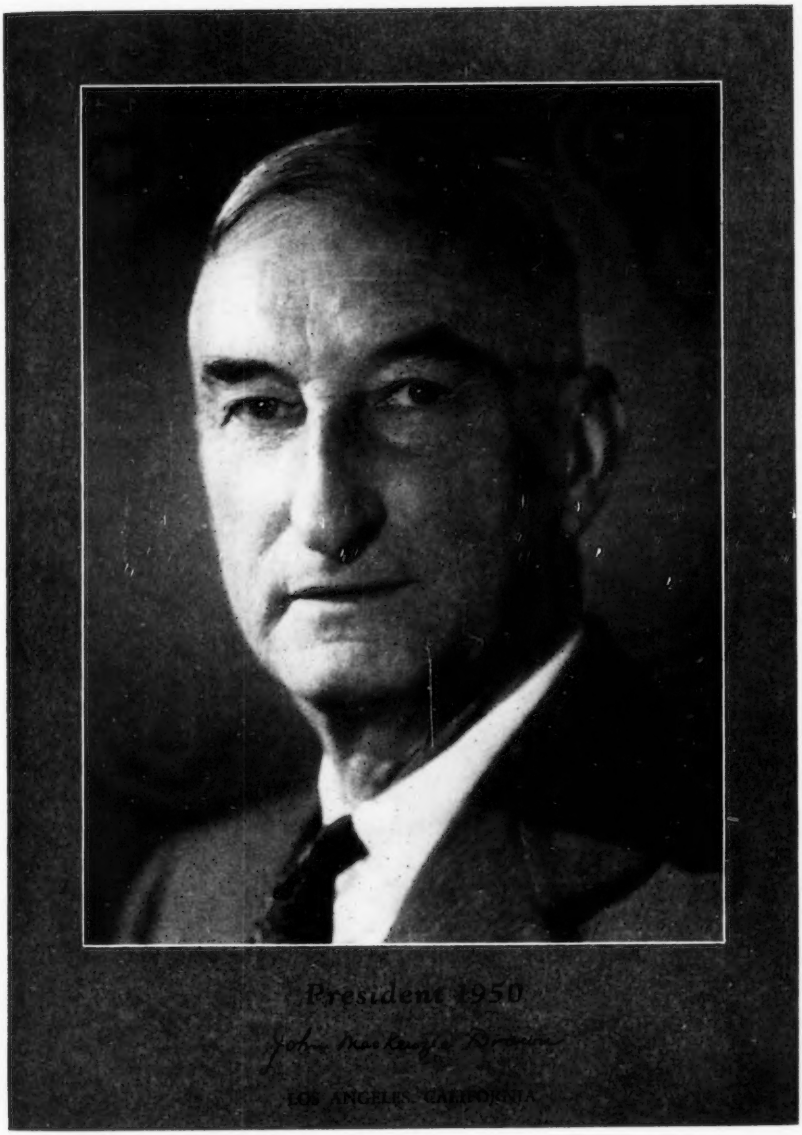
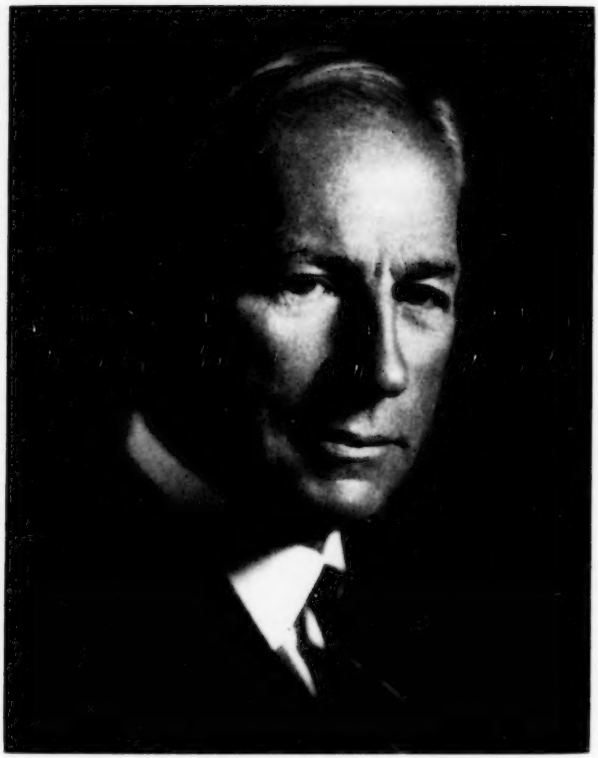


President 1950

John MacKaye Rowan

LOS ANGELES, CALIFORNIA

Guest of Honor



JOHN DEVEREUX KERNAN, M.D.

Graduate of Harvard University, 1900; received his medical degree from Columbia University College of Physicians and Surgeons, 1903. Professor Emeritus of Otolaryngology, Columbia University College of Physicians and Surgeons. Member of the American Medical Association, American Laryngological Association, American Broncho-Esophagological Association, American Association for Thoracic Surgery, American Association of Anatomists, New York Academy of Medicine, New York Laryngological Association, New York State Medical Society, and New York County Medical Society.

FIFTY-FIFTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

OCTOBER 8 TO 13, 1950

PALMER HOUSE

Chicago, Illinois

OFFICIAL NOTICES

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Fifty-Fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Sunday, October 8, 1950, and continue through noon Friday, October 13, 1950.

J. MACKENZIE BROWN, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Local Committees

JOHN R. LINDSAY
General Chairman

Reception Committee

Arlington C. Krause, *Chairman*
John G. Bellows
Paul A. Campbell
James W. Clark
John F. Delph
Justin M. Donegan
Richard C. Gamble
Palmer W. Good
M. R. Guttman
Joseph S. Haas
Jerome W. Hayden
William F. Hughes, Jr.
Peter C. Kronfeld
Francis L. Lederer
Vernon M. Leech
Norman Leshin
Richard E. Marcus
William F. Moncreiff
Herbert T. Nash
W. J. Noonan
Henry B. Perlman
Irving Puntenney
Roy O. Riser
Edward A. Roling
Merton B. Skinner
Daniel Snydacker
O. E. Van Alyea

Committee on Arrangements

Stanton A. Friedberg, *Chairman*
Paul V. Carelli
Carl H. Christoph
Arthur J. Coombs
Maurice H. Cottle
Cyril V. Crane
Clifford L. Dougherty
Noah D. Fabricant
Earle B. Fowler
Orville E. Gordon
Myron M. Hipskind
Louis G. Hoffman
L. J. Lawson
Robert B. Lewy
George S. Livingston
David B. Maher
William A. Mann
Frank W. Newell
Samuel J. Pearlman
Samuel Salinger
Gail R. Soper
Derrick Vail
Linden J. Wallner
Frank Wojniak

Ladies' Committee

Mrs. John R. Lindsay, *Chairman*
Mrs. G. Henry Mundt, *Co-Chairman*
Mrs. Thomas D. Allen
Mrs. Howard C. Ballenger

Mrs. John J. Ballenger
Mrs. Bruno Blumklotz
Mrs. Paul A. Campbell
Mrs. Paul V. Carelli
Mrs. James W. Clark
Mrs. Ralph A. Davis
Mrs. J. Robert Fitzgerald
Mrs. Earle B. Fowler
Mrs. M. M. Hipskind
Mrs. Paul H. Holinger
Mrs. William F. Hughes, Jr.
Mrs. Heinrich Kobrak
Mrs. Peter C. Kronfeld
Mrs. Lawrence J. Lawson
Mrs. Nathan K. Lazar
Mrs. Vernon M. Leech
Mrs. Robert B. Lewy
Mrs. William A. Mann
Mrs. Samuel J. Meyer
Mrs. G. Henry Mundt, Jr.
Mrs. Frank W. Newell
Mrs. S. J. Pearlman
Mrs. Henry B. Perlman
Mrs. Richard A. Perritt
Mrs. Irving Pantenney
Mrs. Kenneth L. Roper
Mrs. Samuel Salinger
Mrs. Harold F. Schuknecht
Mrs. George E. Shambaugh, Jr.
Mrs. Theodore M. Shapira
Mrs. Sherman L. Shapiro
Mrs. Daniel Snydacker
Mrs. Gail R. Soper
Mrs. Clifford P. Sullivan
Mrs. William B. Sullivan
Mrs. Walter H. Theobald
Mrs. Derrick Vail
Mrs. O. E. Van Alyea

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 9 o'clock, Saturday morning, October 7, 1950, in Private Dining Room No. 4 on the third floor of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 11, at 12:45 in Private Dining Room No. 5. Members desiring to be heard or to present business will confer with the Executive Secretary for appointments.

J. MACKENZIE BROWN, M.D.
President

New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Erling W. Hansen, M.D., 90 South Ninth St., Minneapolis 2, Minn., at the earliest possible moment.

Exhibits

The Scientific and Technical Exhibits will open at 9 o'clock Sunday morning, October 8.

Dues

Receipt card for 1950 dues is required for registration at the Academy meeting. Those who have not paid dues for 1950 are requested to remit to W. L. Benedict, M.D., Executive Secretary-Treasurer, 100 First Avenue Bldg., Rochester, Minn. In case of loss, a duplicate receipt card may be obtained upon request.

Registration

General registration will begin at 9 o'clock Sunday morning, October 8, in the Foyer to the Grand Ball Room, Fourth Floor.

The regulation badge secured at the time of registration is essential for admittance to all activities. To facilitate registration, the following sections will be provided:

Advance Orders: Members who have reserved instruction course tickets register here. Advance orders are held here for collection.

Members without Advance Orders: Please present Receipt Card for 1950 dues. Dues outstanding may be paid at this section.
(Application blanks for fellowship in the Academy may be obtained here.)

Instructors: All persons presenting instruction courses register here to secure special badge. There is no registration fee for instructors who are not members.

Non-members: A registration fee of $5.00 is required of all guests (see below). The following exceptions are made:

1. Candidates for Academy fellowship whose names appear in the July-August issue of the TRANSACTIONS.
2. Residents in training and registrants for the 1950-1951 Home Study Courses presenting guest cards obtained before the meeting (see next column).
3. Orthoptic technicians.
4. Guests taking part in the Scientific Programs or Exhibits.

Guests

Physicians who are not members are welcome to attend the meeting of the Academy as guests and to participate in all the activities. All guests, however, will be required to register and a fee of $5.00 will be charged.

In the past, a number of Fellows and registered guests of the Academy have indicated that they wish their *office nurses, technicians,* and *similar personnel* to attend parts of the Academy program or exhibits. Such individuals will be allowed to register upon presentation of a written request from a Fellow or if accompanied by a registered guest or Fellow. The guest fee of $5.00 will be required. An exception is made for orthoptic technicians, who may register without fee.

Residents and 1950-1951 Registrants for Home Study Courses

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, may register without fee upon presentation of a guest card obtained by written request of their Department Heads.

Registrants who have completed enrollment for the 1950-1951 Home Study Courses in Ophthalmology or Otolaryngology may also request a guest card entitling them to registration without fee.

Requests from Department Heads and Home Study Courses registrants for guest cards should be sent to W. L. Benedict, M.D., 100 First Avenue Bldg., Rochester, Minn., by September 30.

Instruction Course Tickets

The tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks in the Registration Headquarters. The bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $1.50 per hour.

It is necessary to have registered and to be wearing regulation badge in order to purchase instruction course tickets.

Ladies' Registration

The wives of members and guests are urged to register at the Ladies' Headquarters in Rooms 15 and 16 on the Club Floor. Registration will open at 9 o'clock, Sunday morning, October 8. Informal coffees and teas will be held on Sunday and Monday.

Arrangements have been made for a tour on Tuesday afternoon of either the Museum of Science and Industry or the University of Chicago Campus. Both groups will have tea at the Quadrangle Club following the tour. Special entertainment will be provided by the University of Chicago Acrotheatre and the Varsity Gymnastic Team of the University of Chicago Physical Education Department.

On Wednesday there will be a luncheon at the Drake Hotel followed by a historical fashion show.

Tickets to these functions, as well as to radio broadcasts, may be obtained at the Ladies' Registration Desk.

Available Supplies

Collected abstracts of instruction courses, manuals and monographs, and bound volumes of the 1949-1950 TRANSACTIONS may be purchased at the supply counter in Registration Headquarters. Guests may place subscriptions to the bimonthly issues of the TRANSACTIONS here.

Dinner Tickets

Tickets for Alumni Dinners and the Annual Academy Dinner will be on sale in the Registration Headquarters. Sale of tickets closes at 2 o'clock in the afternoon of the day of the dinner.

Constitutional Changes in Fellowship Status

Life Fellows. Fellows who have paid dues continuously for thirty years will automatically become Life Fellows. They shall not be required to pay any fees or dues or assessments and shall enjoy all the privileges of Active Fellows.

Senior Fellows. Fellows who have paid dues continuously for twenty-five years will be termed Senior Fellows and their dues will be one-half of the dues of Junior Fellows.

from Article V, Section 6, By-Laws

Life Fellows — 1951

Alden, Arthur M. ..... St. Louis, Mo.
Bahn, Charles A. ..... New Orleans, La.
Brickley, Daniel W. ..... Marion, Ohio
Brown, Mortimer G. ..... Syracuse, N. Y.
Buvinger, Charles W. .... East Orange, N. J.
Cohen, Samuel ..... Philadelphia, Pa.
Darmer, George A. ..... Aurora, Ill.
Dintenfass, Henry ..... Philadelphia, Pa.
Hughes, T. E. ..... Richmond, Va.
Husik, David N. ..... Philadelphia, Pa.
King, Edward Daniel .... Hollywood, Calif.
Lewis, Fielding O. ..... Media, Pa.
Reese, Warren S. ..... Philadelphia, Pa.
Schlanser, Adam E. ..... Washington, D. C.
Williams, Walton H. ..... Middletown, Ohio
Zentmayer, William ..... Philadelphia, Pa.

Senior Fellows — 1951

Beal, Homer A. ..... Kansas City, Mo.
Birsner, Louis J. ..... St. Louis, Mo.
Bonner, William F. ..... Childress, Texas
Brown, Albert L. ..... Cincinnati, Ohio
Chapman, S. Jefferson, Colorado Springs, Colo.
Cleff, Oscar ..... Chicago, Ill.
Daily, Louis ..... Houston, Texas
Daily, Ray K. ..... Houston, Texas
Fox, Noah ..... Chicago, Ill.
Gipner, John F. ..... Rochester, N. Y.
Goar, Everett L. ..... Houston, Texas
Gordon, Charles H. ..... Portland, Maine
Hamlin, Fred E. ..... Roanoke, Va.
Hands, Sidney G. ..... Davenport, Iowa
Hansel, French K. ..... St. Louis, Mo.
Hargitt, Charles A. ..... Brooklyn, N. Y.
Harrell, Voss ..... Reno, Nev.
Hartshorne, Isaac ..... New York, N. Y.
Hicks, Vonnie M. ..... Raleigh, N. C.
Jones, Edmund L. ..... Wheeling, W. Va.
Joy, Harold H. ..... Syracuse, N. Y.
Kettelcamp, Fred O., Colorado Springs, Colo.
Key, Samuel N. ..... Austin, Texas
McLaurin, John G. ..... Dallas, Texas
McMahon, Bernard J. ..... St. Louis, Mo.
Minsky, Henry ..... New York, N. Y.
Morrison, W. Wallace .... New York, N. Y.
Myers, E. Lee ..... St. Louis, Mo.
Packard, Louis A. ..... Phoenix, Ariz.
Palmer, Arthur ..... New York, N. Y.
Pember, Aubrey H. ..... Janesville, Wis.
Pendexter, R. Stevens .... Washington, D. C.
Reineke, George F. ..... New Ulm, Minn.
Seal, J. Coleman ..... New York, N. Y.
Seligstein, Milton B. ..... Memphis, Tenn.
Sharp, Benjamin S. ..... Providence, R. I.
Stokes, William H. ..... Lake City, Mich.
Wagener, Henry P. ..... Rochester, Minn.
Wright, Walter W. .... Toronto, Ont., Can.
Young, Charles A. ..... Roanoke, Va.

BUSINESS MEETING

Crystal Room

Thursday, October 12, 1950

5:30 p.m.

ORDER OF BUSINESS

1. Call to order.
2. Report of Executive Secretary-Treasurer.
3. Report of the Senior Member of the Council.
4. Election of Fellows.
   (See Candidate list below.)
5. Election of officers.
6. Unfinished business.
7. New business.

CANDIDATES — 1950

Objections to the election of a candidate must be filed with the Executive Secretary previous to Saturday, October 7, 1950.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (OP)
Otolaryngology (ALR)
Plastic Surgery (Pl S)

Acquarelli, Mario John, Wadsworth General Hospital, Los Angeles 25, Calif., ALR.

Albers, George Donald, 8½ Monroe St., Grand Rapids 2, Mich., ALR.

Anderson, Elam DeMar, Medical & Dental Bldg., Seattle 1, Wash., OP.

Anderson, Elbert Carl, 201 N. Front St., Wilmington, N. C., OP.

Anderson, H. Gordon, 403 Fulton St., Troy, N. Y., OP.

Anslow, Robert Elmer, 10 Peterboro St., Detroit 1, Mich., OP.

Baers, Harry Arnold, 4063 Radford Ave., Studio City, Calif., ALR.

Baldridge, Max N., P.O. Box 778, Texarkana, Ark.-Texas, OP.

Barbee, John Young, 1109 State St., Bowling Green, Ky., ALR.

Barrere, Luciano Enrique, Tulane University, New Orleans 12, La., OP.

Barton, William Lawrence, 403 Persons Bldg., Macon, Ga., ALR.

Bergman, Macks Leonard, 8443 Crenshaw Blvd., Inglewood, Calif., ALR.

Birmingham, Eugene E., 333 W. North Ave., Chicago 10, Ill., ALR.

Blair, James Robert, 920 Metropolitan Bldg., Denver 2, Colo., ALR.

Blanford, Sidney Edgar, Jr., 1624 Gilpin St., Denver, Colo., Pl S.

Blodi, Frederick C., 635 W. 165th St., New York 32, N. Y., OP.

Bloomberg, Louis, 506 Central Tower Bldg., Youngstown 3, Ohio, OP.

Bobbett, Gordon Howard, 125 W. Cheves St., Florence, S. C., ALR.

Boshnack, Malcolm, 70 Strawberry Hill Ave., Stamford, Conn., ALR.

Bosworth, Wesley F., E. Main St., Clarinda, Iowa, OP.

Boucher, Irvan Andrew, 1221 12th Ave., Altoona, Pa., ALR.

Braveman, Bernard Leon, 412 Fifth Ave., McKeesport, Pa., OP.

Breffeilh, Louis Andrew, 421 Medical Arts Bldg., Shreveport, La., OP.

Brown, Kenneth Brien, 131 Fulton Ave., Hempstead, N. Y., ALR.

Bryan, John Thomas, 2105 Hayes St., Nashville 5, Tenn., ALR.

Burnham, Charles Joseph, 1529 N. 25th St., Birmingham, Ala., OP.

Burr, Sherwood Petersen, 43 E. Jackson St., Tucson, Ariz., OP.

Butler, Jay B. V., 919 Taylor Street Bldg., Portland 5, Ore., OP.

Cairns, Adrian Bennett, 415 Pere Marquette Bldg., New Orleans 12, La., ALR.

Callaghan, Winship C., Union Trust Bldg., Greensburg, Ind., ALR.

Callahan, Neil, 315 Medical Arts Bldg., Norfolk 10, Va., ALR.

Cammack, Bragg Charles, 6381 Hollywood Blvd., Hollywood 28, Calif., ALR.

Capriotti, Octavius A., 404 E. Broad St., Souderton, Pa., OP.

Carris, James Vernon, 209 S. Nevada Ave., Colorado Springs, Colo., ALR.
Casey, Edwin Joseph, 508 N. Grand Ave., St. Louis 3, Mo., OP.
Christenberry, Kenneth William, 501 W. Church Ave., Knoxville, Tenn., OP.
Clark, Archibald Fletcher, Jr., 225 Medical Arts Bldg., San Antonio 5, Texas, ALR.
Clay, Richard A., 416 N.W. 13th St., Oklahoma City, Okla., OP.
Clayton, Sam, 119-05 80th Road, Kew Gardens 15, N. Y., ALR.
Coleman, Howe Reese, 305 Young Bldg., Lynchburg, Va., ALR.
Corgill, Donald Alton, Veterans Administration Hospital, McKinney, Texas, ALR.
Crane, Edward Harrison, Jr., 127 S. Grevillea Ave., Inglewood, Calif., ALR.
Crawford, Walter James, 3333 Pachappa Drive, Riverside, Calif., OP.
Cressman, Frederic E., 102 S. Second St., Artesia, N. M., OP.
Crowder, Miles S., 603 W. Main St., Knoxville, Tenn., OP.

Daly, Joseph M., 1801 K St. N.W., Washington 6, D. C., ALR.
DeLuca, Charles Q., 255 S. 17th St., Philadelphia 3, Pa., ALR.
Denicke, Ernest Webber, 1010 B St., San Rafael, Calif., OP.
Dennis, Richard Hollis, 33 College Ave., Waterville, Maine, OP.
Dillahunt, Jack A., Copper Ave. at Monroe, Albuquerque, N. M., OP.
Dorman, Purman, 1215 Fourth Ave., Seattle 1, Wash., OALR.
Dryden, James Spencer, 1835 Eye St. N.W., Washington 6, D. C., OP.
Duncan, Herbert, 700 Church St., Nashville 3, Tenn., ALR.
duPrey, Robert E., 1150 Connecticut Ave., Washington 6, D. C., OP.
Dwyer, Gregory Kennedy, Community Medical Group, Boonton, N. J., ALR.

Edelstein, Isidore S., 259 New York Ave., Brooklyn 16, N. Y., OP.
Edwards, Thomas Luther, 507 S. Washington St., Van Wert, Ohio, OP.
Egdorf, Otto Charles, 420 Hamilton Bldg., Wichita Falls, Texas, ALR.
Esbin, Leo, 1333 President St., Brooklyn 13, N. Y., OP.
Esposito, Albert Charles, First Huntington National Bank Bldg., Huntington 1, W. Va., OP.
Eubank, William Richards, 1102 Grand Ave., Kansas City 6, Mo., OP.
Evans, John W., 325 Franklin St., Huntsville, Ala., ALR.

Failla, Anthony, 3831 Frenchmen St., New Orleans, La., ALR.
Fairbanks, Stephen, 119½ N. Superior St., Albion, Mich., OP.
Feher, Alexander, 123 E. 83rd St., New York 28, N. Y., ALR.
Feldman, John L., 311 Division St., Quincy, Ill., ALR.
Feldstein, Morris, 515 Park Ave., New York 22, N. Y., OP.
Fields, James Allan, Naval Hospital, Beaufort, S. C., ALR.
Florentz, Theodore Robert, 109 N. Eighth St., Boise, Idaho, OP.
Follette, William James, 375 Engle St., Englewood, N. J., ALR.
Friedman, Bernard B., 916 Jones Bldg., Corpus Christi, Texas, OP.
Friedman, Isadore Edward, 5248 Hohman St., Hammond, Ind., ALR.
Friedman, Paul Norman, 1111 St. Paul St., Baltimore 2, Md., OP.
Fuchs, Jesse, 6363 Wilshire Blvd., Los Angeles 48, Calif., ALR.

Gardner, James Frank, 11 N. Goodman St., Rochester, N. Y., ALR.
Garner, Lawrence Lee, 238 W. Wisconsin Ave., Milwaukee 3, Wis., OP.
Garron, Levon K., 426 17th St., Oakland 12, Calif., OP.
Gifford, Edward Stewart, Jr., 1913 Spruce St., Philadelphia 3, Pa., OP.
Gillman, A. Marvin, 54 E. 72nd St., New York 21, N. Y., OP.
Girard, Louis Joseph, Centre Island, Long Island, N. Y., OP.
Glass, Walter Martin, 221 Middle Neck Rd., Great Neck, N. Y., ALR.
Godwin, Robert William, 117 E. Eighth St., Long Beach 13, Calif., ALR.
Golden, Samuel Charles, 150 Prichard St., Fitchburg, Mass., ALR.
Goldsmith, Charles Porter, 1648 Hamilton St., Allentown, Pa., OP.
Gooch, J. Oliver, 1203 Hermann Professional Bldg., Houston 5, Texas, ALR.
Gorin, George, 885 West End Ave., New York 25, N. Y., OP.
Grayman, Harry Myer, 2900 Fresno Street Bldg., Fresno 1, Calif., OP.
Greene, Richard W., 32 Johnson Ave., Newark 8, N. J., OP.
Greenfield, S. Jerome, 31 Lincoln Park, Newark 2, N. J., OP.

Greer, Rex E., 308 N. Colorado. Midland, Texas, ALR.
Gros, Jose Conrad, Calle 25 no. 960, Vedado, La Habana, Cuba, ALR.
Guida, Francis Paul, 67 Trumbull St., New Haven 10, Conn., OP.

Haight, Whitney James, 9 Exchange Place, Salt Lake City 1, Utah, ALR.
Harms, Edwin M., 104 W. Douglas St., Wichita 2, Kan., OP.
Harper, Donald McCarthy, 1915 New Hampshire Ave., N.W., Washington 9, D. C., ALR.
Hatfield, Haskell Doke, 1201 First National Bldg., El Paso, Texas, ALR.
Hathcock, William Caldwell, 402 Grand Theatre Bldg., Atlanta 3, Ga., OP.
Heck, Walter Emil, Stanford University Hospital, San Francisco, Calif., ALR.
Heller, Morris Freund, 115 E. 61st St., New York 21, N. Y., ALR.
Herman, Seymour J., 1601 E. 23rd St., Brooklyn 29, N. Y., ALR.
Hirst, William Randolph, 2241 Central Ave., Alameda, Calif., OP.
Hoffman, Franklin David, 626 Union Trust Bldg., Pittsburgh 19, Pa., OP.
Hoffman, Parker M., 134 E. First St., Corning, N. Y., OP.
Hogg, Stephen P., 556 Doctors' Bldg., Cincinnati 2, Ohio, ALR.
Hosner, James Wesley, 255 S. 17th St., Philadelphia 3, Pa., OP.
Hull, Forrest Edgar, Tokyo General Hospital, APO 1052, c/o PM, San Francisco, Calif., OP.
Huston, James M., 1280 E. 14th St., San Leandro, Calif., ALR.
Hynes, Edward Allen, U. S. Naval Academy, Annapolis, Md., OP.
Hyslop, Volney Butman, 759 N. Milwaukee St., Milwaukee 2, Wis., Pl S.

Ittkin, Paul, 5921 Clark St., Montreal, Que., Canada, ALR.

Jennings, Edward C., 2650 Wisconsin Ave., Washington 7, D. C., ALR.

Kant, Alfred, 129 Clinton St., Watertown, N. Y., OP.
Katz, Jacob, 1807 S. Sixth St., Philadelphia 48, Pa., OP.
Keck, William Struble, 122 Main St., Greensburg, Pa., ALR.
Kelemen, George, 20 Gloucester St., Boston 15, Mass., ALR.
Kennon, William G., Jr., 706 Church St., Nashville 3, Tenn., ALR.
Kimmelman, David Brown, 10 Downing St., New York 14, N. Y., OP.
Kirsch, Ralph Emile, 903 Huntington Bldg., Miami 32, Fla., OP.
Kirshner, Harold, 20 Park Ave., New York 16, N. Y., OP.
Knapp, Philip, University Hospitals, Iowa City, Iowa, OP.
Kolodny, George Robert, 702 Hermann Professional Bldg., Houston 5, Texas, OP.
Krishna, Ikbal, 1049 E. Washington St., Brownsville, Texas, ALR.
Krug, Joseph Hoffmann, 988 Fifth Ave., New York 21, N. Y., OP.
Kunkel, William Howard, 308 Medical Arts Bldg., Pittsburgh 13, Pa., ALR.

Lane, Charles Spurgeon, Jr., 205 Merchants National Bank Bldg., Fort Smith, Ark., ALR.
Lateiner, Robert, 650 Main St., New Rochelle, N. Y., OP.
Latella, Peter D., 524 North Ave., New Rochelle, N. Y., ALR.
Lauren, George Peter, 205 Medico-Dental Bldg., San Diego 1, Calif., OP.
Lavoie, Roland, 3 Claire-Fontaine, Quebec City, Que., Canada, ALR.
Lebo, Charles Phillip, 655 Sutter St., San Francisco 2, Calif., ALR.
Lee, Jack Bennett, 607 New Moore Bldg., San Antonio 5, Texas, OP.
Lewis, Paul M., 519 N. Highland Ave., Pittsburgh 6, Pa., ALR.
Lhotka, Frank M., 6005 Cermak Road, Cicero 50, Ill., OP.
Liebman, Sumner David, 115 Bay State Road, Boston 15, Mass., OP.
Linden, Arthur Joseph, Shirlington Theatre Bldg., Arlington, Va., ALR.
Locklin, Walter Kaye, 1410 American National Bank Bldg., Kalamazoo, Mich., ALR.
Loring, Milton Jack, 304 North 'N' St., Midland, Texas, OP.
Lovely, David K., 73 Deering St., Portland 4, Maine, ALR.
Lowrey, Austin, Walter Reed Hospital, Washington 12, D. C., O.P.
Loy, David Taylor, 3207 W. 16th St., Great Bend, Kan., OP.
Luedde, Fullerton Woods, 35 N. Central Ave., St. Louis (Clayton) 5, Mo., OP.
Lymberis, Marvin Nicholas, 106 W. Seventh St., Charlotte 2, N. C., OP.

MacMillan, Charles Wright, 4 Duryea Road, Upper Montclair, N. J., ALR.

Magnet, Isaac Harry, 130 Rock St., Fall River, Mass., ALR.
Maloney, Walter Hugh, Green and Coulter Sts., Philadelphia 44, Pa., ALR.
Mandelbaum, Joseph, 101 Lafayette Ave., Brooklyn 17, N. Y., OP.
Mannis, Aaron A., 109 N. Wabash Ave., Chicago 2, Ill., OP.
Marshall, Alexander Washington, 145-A Rutledge Ave., Charleston 17, S. C., OP.
Mathes, William Thomas, Jr., Jones EENT Hospital, Johnson City, Tenn., ALR.
McFarland, James Jerry, 1150 Connecticut Ave., N.W., Washington 6, D. C., ALR.
McGee, Hugh E., 117 Cedar Lane, Teaneck, N. J., OP.
McGowan, William Louis, Union Central Bldg., Cincinnati 2, Ohio, OP.
McKeigue, John E., 94 Pleasant St., Arlington, Mass., ALR.
Medof, Milton I., 130 S. Robertson Blvd., Beverly Hills, Calif., ALR.
Merriam, George Rennell, Jr., 635 W. 165th St., New York 32, N. Y., OP.
Merz, Arthur Edgar, 821 Franklin Ave., Garden City, N. Y., OP.
Meyer, Eugene A., 427 Cooper St., Camden 2, N. J., ALR.
Michelson, Robin P., 240 Stockton, San Francisco 8, Calif., ALR.
Mietus, Conrad A., 930 Fillmore Ave., Buffalo 11, N. Y., OP.
Milanese, Nicholas Peter, 123 E. 83rd St., New York 28, N. Y., ALR.
Miller, Jack Barnett, 4212 N.E. Broadway, Portland 13, Ore., ALR.
Miller, William Jacob, 21 E. State St., Columbus, Ohio, ALR.
Minnes, James Fortin, 1701 W. Broadway, Vancouver, B. C., Canada, OP.
Mishler, Jay Eli, 1616 Pacific Ave., Atlantic City, N. J., OP.
Mitchell, Howard Lysle, 103 S. Main St., Lexington, Va., ALR.
Montgomery, Earl Clifton, 1620 Medical Arts Bldg., Omaha 2, Neb., ALR.
Moore, Ralph Lewis, 509 N. Broad St., Woodbury, N. J., ALR.
Moorman, Victor Reuben, Wiley Bldg., Hutchinson, Kan., ALR.
Moreland, Joseph Ivan, 2485 Center St., Salem, Ore., OP.
Morgenstern, David Jacob, 433 Eastern Parkway, Brooklyn 16, N. Y., ALR.
Morrison, Lewis E., 503 Hume Mansur Bldg., Indianapolis 4, Ind., ALR.
Moulton, Everett Crockett, Jr., 205 Merchants National Bank, Fort Smith, Ark., OP.
Mulberger, Robert D., 1930 Chestnut St., Philadelphia 3, Pa., OP.
Myers, Roland Horace, 1720 Exchange Bldg., Memphis 3, Tenn., OP.

Nakashima, Victor Katsuhiro, Veterans Administration, Des Moines 10, Iowa, ALR.
Nickeson, Robert Warren, 179 Allyn St., Hartford 3, Conn., OP.
Nisbet, Alfred Alan, 1110 South Texas Bldg., San Antonio, Texas., OP.
Noble, Bertha Riveroll, 1430 Tulane Ave., New Orleans 12, La., OP.

O'Connell, John D., 50 Farmington Ave., Hartford 5, Conn., OP.
Olson, James Albert, Henry Ford Hospital, Detroit 2, Mich., OP.
O'Neill, John Campbell, 205 W. Second St., Duluth, Minn., OP.
Orzac, Edward Seymour, 460 Rockaway Ave., Valley Stream, N. Y., ALR.

Parker, Francis William, 1102 Broadway, Rockford, Ill., OP.
Parks, Kirtland Garvin, 605 Professional Bldg., Long Beach 13, Calif., OP.
Paul, Thomas Otis, 2205 Highland Ave., Birmingham 5, Ala., OP.
Pedersen, Paul Milton, 2241 Central Ave., Alameda, Calif., ALR.
Pendexter, Sidney Eugene, Jr., 11 S. Arlington Ave., East Orange, N. J., OP.
Perzia, Anthony Peter, 910 Citizens Bldg., Tampa 2, Fla., OP.
Peterson, John Hartley, 812 Medical Arts Bldg., Duluth, Minn., OP.
Plotke, Harry L., 1058 Lowry Medical Arts Bldg., St. Paul 2, Minn., OP.
Pole, Samuel Boyce, III, 200 W. Commerce St., Bridgeton, N. J., OP.
Polisar, Ira Allan, 142 Joralemon St., Brooklyn 2, N. Y., ALR.
Powell, James Robert, 501 Medico-Dental Bldg., Stockton, Calif., OP.
Proctor, Malvin, 66 Main St., Tuckahoe 7, N. Y., OP.

Rackwitz, George, 245 E. Broadway, New York 2, N. Y., OP.
Rambo, John Henry T., 119 E. 74th St., New York 21, N. Y., ALR.
Rea, Robert P., Gorgas Hospital, Ancon, Canal Zone, ALR.

Reagan, Daniel Joseph, 507 Main St., Worcester 8, Mass., OP.
Reddy, John Bernard, U. S. Naval Hospital, Annapolis, Md., ALR.
Reitz, Russell E., 446 Linwood Ave., Buffalo 9, N. Y., OP.
Reynolds, Garland Alexander, 826 Themis St., Cape Girardeau, Mo., ALR.
Richardson, George S., 201 S. Arno St., Albuquerque, N. M., ALR.
Richardson, James Mark, 6306 Cottage Grove Ave., Chicago 37, Ill., OP.
Richardson, Oscar B., 880 Bay St., Toronto 5, Ont., Canada, OP.
Rockwell, Albert George, Jr., 300 Homer Ave., Palo Alto, Calif., ALR.
Romano, John Emil, 4010 W. Madison, Chicago 24, Ill., ALR.
Rooker, Richard W., 423 Walnut Ave., Niagara Falls, N. Y., ALR.
Ross, Maurice E., 535-E Grand, Beloit, Wis., ALR.
Rothman, Harold, 41 Eastern Parkway, Brooklyn 17, N. Y., OP.
Russell, William Marler, New Bank Asheville Bldg., Asheville, N. C., ALR.
Ryan, Robert Emmett, 3903 Olive St., St. Louis 8, Mo., ALR.
Sacks-Wilner, Erwin Preston, 225 W. State St., Trenton 8, N. J., OP.
Sanderson, Bruce A., 2575 E. Eighth St., National City, Calif., ALR.
Santamarina, Fernando Garcia, Hotel North Park, Chicago 14, Ill., ALR.
Saunders, Joseph Hamilton, 288 S. Limestone St., Lexington, Ky., OP.
Schiff, Maurice, 5459 Diamond St., Philadelphia 3, Pa., ALR.
Schillinger, Robert John, 727 W. Seventh St., Los Angeles 14, Calif., OP.
Schuknecht, Harold F., 950 E. 59th St., Chicago 37, Ill., ALR.
Schutz, William Jack, 672 Francis Bldg., Louisville 2, Ky., OP.
Shafer, Donald McKay, 140 E. 54th St., New York 22, N. Y., OP.
Shaffer, Robert Nesbit, 490 Post St., San Francisco 2, Calif., OP.
Shepherd, Edwin McRae, 1106 Virginia St., Charleston 1, W. Va., OP.
Sherman, Henry Knapp, 121 University Place, Pittsburgh 13, Pa., ALR.
Shofstall, William Howard, 300 W. 47th St., Kansas City, Mo., ALR.
Simmons, Frederick H., U. S. Veterans Administration Hospital, Marion, Ind., ALR.
Simses, John P., 144 Golden Hill St., Bridgeport 3, Conn., OP.
Skolnik, Emanuel Mitchell, 55 E. Washington St., Chicago 2, Ill., ALR.
Smith, Graham Gable, 304 Doctors' Bldg., Minneapolis 2, Minn., ALR.
Smith, Hal Waugh, Second & "G" Sts., San Rafael, Calif., ALR.
Smith, Joseph George, 490 Post St., San Francisco 2, Calif., OP.
Smith, Trent W., 345 E. State St., Columbus, Ohio, ALR.
Spencer, James Avery, Lettunich Bldg., Watsonville, Calif., OP.
Spencer, James Thomas, Jr., 1112 Virginia St., E., Charleston 1, W. Va., ALR.
Spiro, Barbara, 7449 Cottage Grove Ave., Chicago 19, Ill., OP.
Springer, Kurt C., 504 Arcade Bldg., Kankakee, Ill., ALR.
Stack, David Rodney, Jr., 641 David Whitney Bldg., Detroit 26, Mich., ALR.
Stancil, James Rose, Medical Dental Center Bldg., Bellingham, Wash., ALR.
Stanfill Charles Mac, Medical Arts Bldg., El Paso, Texas, ALR.
Steffensen, Ellis H., Henry Ford Hospital, Detroit 2, Mich., OP.
Steiner, Albert, 1308 Eutaw Place, Baltimore 17, Md., ALR.
Stillerman, Manuel Leon, 109 N. Wabash Ave., Chicago 2, Ill., OP.
Stone, Vean Melford, 3616 Main St., Riverside, Calif., OP.
Stonehill, Alfred A., 6 N. Michigan Ave., Chicago 2, Ill., OP.
Stuart, Edwin Alexander, 1390 Sherbrooke St., W., Montreal, Que., Canada, ALR.
Sun, Kuei Shu, 950 E. 59th St., Chicago 37, Ill., OP.

Tabb, Harold Granberry, 1124 Maison Blanche Bldg., New Orleans 16, La., ALR.
Taylor, George Dekle, 111 W. Adams St., Jacksonville, Fla., ALR.
Teitgen, Ralph Emil, Mayo Clinic, Rochester, Minn., OP.
Thompson, Floyd Forrest, 1301 N. Broadway, Santa Ana, Calif., ALR.
Thumim, Mark, 121 Main St., Middletown, Conn., ALR.
Tibbetts, Otis Benson, 33 Court St., Auburn, Maine, OP.
Titche, Leon L., Veterans Administration Hospital, Tucson, Ariz., ALR.
Trent, Robert Irvine, 708 Medical Arts Bldg., Oklahoma City 2, Okla., OP.
Trombetta, Alessandro, U. S. Naval Hospital, San Diego 34, Calif., ALR.

Trotter, John H., 212 High St., Morgantown, W. Va., OP.
Turley, John C., 899 Madison, Memphis, Tenn., ALR.

Ulvestad, Harold S., 202 Doctors' Bldg., Minneapolis 2, Minn., ALR.
Underwood, Ernest Arthur, 601 Main St., Vancouver, Wash., OP.

Virant, John A., 906 Olive St., St. Louis 1, Mo., ALR.
Voorhees, Charles Hammell, 308 College Ave., Elmira, N. Y., OP.

Wainstock, Michael Allen, 1508 David Broderick St., Detroit 26, Mich., OP.
Walker, Donald H., Dollar Title Bldg., Sharon, Pa., ALR.
Walker, James S., 602 W. University Ave., Urbana, Ill., ALR.
Waters, Zack James, 220 Camden Ave., Salisbury, Md., ALR.
Weih, Jack E., University Hospitals, Iowa City, Iowa, OP.
Weinstein, Francis Saul, 840 S. 11th St., Newark 8, N. J., ALR.
Weisman, Edward, 2336 Coney Island Drive, Brooklyn 23, N. Y., OP.
Wells, Aubrey Hanson, 400 Hart-Albin Bldg., Billings, Mont., ALR.
West, George Brooks, Jr., 9 Race St., Cambridge, Md., ALR.
West, Stephen Lewis, 5228 Woodlawn Ave., Chicago 15, Ill., ALR.
Westsmith, Richard Alan, 30 S. El Camino Real, San Mateo, Calif., OP.
Wexler, Manuel R., 1917 Wilshire Blvd., Los Angeles 5, Calif. ALR.
Whitaker, Charles Frederic, 55 E. Washington St., Chicago 2, Ill., ALR.
White, Irving Leonard, 812 Pine Ave., Long Beach 13, Calif., ALR.
Wiesenthal, Fred, 30-77 36th St., Astoria, N. Y., OP.
Wiesinger, Warren Edward, 3022 E. 14th St., Oakland, Calif., ALR.
Wolff, Joachim Berthold, 125 E. 84th St., New York 28, N. Y., OP.
Wolkowicz, Michal I., 2959 Richmond St., Philadelphia 34, Pa., OP.
Wright, Joseph William, Jr., 301 Hume-Mansur Bldg., Indianapolis 4, Ind., ALR.

Zurik, Samuel, 3706 Prytania St., New Orleans 12, La., ALR.

CANDIDATES NOT CERTIFIED

(Eligible for Membership After Board Certification)

Beasley, Clifton Harold, 1216 Pennsylvania Ave., Fort Worth, Texas.
Freeman, David M., McMillan Hospital, St. Louis 12, Mo.
Hoch, Carl William, University Hospitals, Iowa City, Iowa.
Love, William Robert, 421 Main St., E., Hamilton, Ont., Canada.
Scheer, Alan Austin, 522 West End Ave., New York 24, N. Y.
Shier, Julius Milton, 585 Main Ave., Passaic, N. J.
Thornhill, Gabriel Felder, 1304 Capital National Bank Bldg., Austin, Texas.

CONVENTION ACTIVITIES

PRESIDENT'S RECEPTION

Grand Ball Room

Sunday, 4:30 p.m.

President J. Mackenzie Brown invites all members and guests to a reception honoring Dr. John D. Kernan and officers of the Academy.

WHERRY MEMORIAL LECTURE

Sponsored by the Wherry Memorial Fund

Dr. Kenneth M. Day, Pittsburgh, Pa., will deliver the Wherry Memorial Lecture at the scientific session of the Section on Otolaryngology, Wednesday morning, October 11. His subject will be "The Management of Deafness."

JACKSON MEMORIAL LECTURE

Sponsored by the Ophthalmic Publishing Co.

Dr. Frank B. Walsh, Baltimore, Md., will deliver the Jackson Memorial Lecture at the scientific session of the Section on Ophthalmology, Wednesday afternoon, October 11. His subject will be "Optic Nerve Sheath Hemorrhage."

SMOKER

Grand Ball Room

Monday, 9:00 p.m.

The ladies are invited. Entertainment, refreshments and music for dancing will be provided.

ANNUAL ACADEMY DINNER

Grand Ball Room

Wednesday, 7:00 p.m.

Dress Optional

Dr. J. Roscoe Miller, president of Northwestern University, Evanston, Ill., will address the assembled guests.

FACULTY LUNCHEON

Private Dining Room No. 14

Thursday, 12:45 p.m.

President J. Mackenzie Brown will be host to all those presenting scientific papers, motion pictures, scientific exhibits, and courses of instruction.

ANNUAL BUSINESS MEETING

Crystal Room

Thursday, 5:30 p.m.

All Fellows and candidates for election to fellowship are urged to attend.

ALUMNI DINNERS

Monday, 6:30 p.m.

Dr. Francis Heed Adler Alumni
Location to be announced
Francis Heed Adler, M.D., Secretary
313 S. 17th St.
Philadelphia 3, Pa.

Baltimore Eye, Ear and Throat Resident Association
Morrison Hotel
Paul N. Friedman, M.D., Secretary
1111 St. Paul St.
Baltimore 2, Md.

Bellevue Hospital Alumni
Palmer House
Private Dining Room No. 6
W. W. Wilkerson, Jr., M.D., Secretary
713 Bennie Dillon Bldg.
Nashville 3, Tenn.

Boston City Hospital Alumni
Stevens Hotel
Private Dining Room No. 6
Benjamin Riseman, M.D., Secretary
41 Bay State Rd.
Boston 15, Mass.

Brooklyn Eye and Ear Hospital Alumni
Palmer House
Private Dining Room No. 5
Leo J. Croll, M.D., Secretary
1326 Maccabees Bldg.
Detroit 2, Mich.

Dr. J. Mackenzie Brown Alumni
Palmer House
Private Dining Room No. 2
Alden H. Miller, M.D., Secretary
500 S. Lucas Ave.
Los Angeles 17, Calif.

Charity Hospital Alumni
Morrison Hotel
Nolley C. Farrington, M.D., Secretary
1407 S. Carrollton Ave.
New Orleans, La.

University of Chicago—Billings Hospital Alumni
Cliff Dwellers Club—Dining Room
(220 S. Michigan Ave.)
Jack P. Cowen, M.D., President
122 S. Michigan Ave.
Chicago 3, Ill.

Cleveland Clinic Alumni
La Salle Hotel
Donald R. Brumley, M.D., Secretary
225 W. Sandusky St.
Findlay, Ohio

Cook County Hospital Alumni
Stevens Hotel
Private Dining Room No. 2
Paul V. Carelli, M.D., Secretary
3626 W. Chicago Ave.
Chicago 51, Ill.

Emory University Alumni
(Former staff members)
Palmer House
Empire Room
(Members will meet at 6:00 p.m. in the room of Dr. Alton V. Hallum)
Wm. Jerome Knauer, M.D., Secretary
206 W. Forsyth St.
Jacksonville 2, Fla.

Episcopal Hospital Residents' Association
La Salle Hotel
E. S. Caldemeyer, M.D., Secretary
1826 R St. N.W.
Washington 9, D. C.

Gill Memorial Eye, Ear and Throat Hospital—Annual Spring Graduate Course Alumni
Palmer House
Illinois Room
E. G. Gill, M.D., Secretary
711 S. Jefferson St.
Roanoke, Va.

Dr. John F. Gipner Alumni
Stevens Hotel
Private Dining Room No. 5
Dewey D. Yoder, M.D., Secretary
725 Washington St.
Columbus, Ohio

Dr. Harry S. Gradle—Michael Reese Hospital Alumni
Standard Club
Theodore M. Shapira, M.D., Secretary
109 N. Wabash Ave.
Chicago 2, Ill.

Harper Hospital Alumni
Palmer House
Private Dining Room No. 3
Wesley G. Reid, M.D., Secretary
974 Fisher Bldg.
Detroit 2, Mich.

Illinois Eye and Ear Infirmary Alumni
Palmer House
Private Dining Room No. 14
Earl S. McRoberts, M.D., Secretary
55 E. Washington St.
Chicago 2, Ill.

University of Illinois Alumni—Otolaryngology
Morrison Hotel
Embassy Room
Noah D. Fabricant, M.D., Secretary
1853 W. Polk St.
Chicago 12, Ill.

Iowa University Alumni
Palmer House
Private Dining Room No. 4
Byron M. Merkel, M.D., Secretary
1112 Equitable Bldg.
Des Moines 9, Iowa

Dr. Chevalier Jackson Alumni
La Salle Hotel
C. L. Jackson, M.D., Secretary
3401 N. Broad St.
Philadelphia, Pa.

Dr. John R. Lindsay Alumni
Stevens Hotel
M. M. Hipskind, M.D., Secretary
6060 Drexel Ave.
Chicago, Ill.

Manhattan Eye, Ear and Throat Hospital Ex-House Surgeons
La Salle Hotel
Parlor D, Mezzanine Floor
W. F. Hubble, M.D., Secretary
268 S. Westlawn Ave.
Decatur, Ill.

Massachusetts Eye and Ear Infirmary Alumni—Ophthalmology
Stevens Hotel
Private Dining Room No. 1
Albert N. Lemoine, Jr., M.D., Secretary
411 Alameda Rd.
Kansas City, Mo.

Massachusetts Eye and Ear Infirmary and Harvard Medical School Alumni—Otolaryngology
Palmer House
Private Dining Room No. 9
August L. Beck, M.D., Secretary
421 Huguenot St.
New Rochelle, N. Y.

Mayo Clinic Alumni
Palmer House—7th Floor
M. E. Nesbit, M.D., Secretary
1 S. Pinckney
Madison, Wis.

McCaskey-Barnhill Alumni
Palmer House
Private Dining Room No. 7
C. H. McCaskey, M.D., Secretary
608 Guaranty Bldg.
Indianapolis 4, Ind.

Memphis Eye, Ear, Nose and Throat Hospital and John Gaston Hospital Alumni
Stevens Hotel
Private Dining Room No. 10
Charles M. King, M.D., Secretary
899 Madison Ave.
Memphis 3, Tenn.

Minnesota University Alumni
Palmer House
Private Dining Room No. 17
Francis M. Walsh, M.D., Secretary
3939 W. 50th St.
Minneapolis 10, Minn.

New York Eye and Ear Infirmary Alumni
Palmer House
Crystal Room
Hunter H. Romaine, M.D., Secretary
218 Second Ave.
New York, N. Y.

New York University Post-Graduate Medical School Alumni
Palmer House
Private Dining Room No. 8
James W. Smith, M.D., Secretary
1016 Fifth Ave.
New York 28, N. Y.

Walter R. Parker—R. Bishop Canfield Alumni
(Former staff members)
Palmer House
Private Dining Room No. 11
Harold F. Falls, M.D., Secretary
University Hospital
Ann Arbor, Mich.

Pennsylvania University and Wills Hospital Alumni—Ophthalmology
University Club
Boulevard Room
(Monroe and Michigan)
Virgil Wescott, M.D., Secretary
30 N. Michigan Ave.
Chicago 2, Ill.

Pennsylvania University Alumni — Otolaryngology
Palmer House
Private Dining Room No. 18
Oram R. Kline, M.D., Secretary
514 Cooper St.
Camden, N. J.

Pittsburgh Eye and Ear Group
Morrison Hotel
Bernard L. Silverblatt, M.D., Secretary
3401 Fifth Ave.
Pittsburgh, Pa.

Presbyterian Hospital Institute of Ophthalmology Alumni
Palmer House
Private Dining Room No. 1
Robert R. Chace, M.D., Secretary
635 W. 165th St.
New York 32, N. Y.

Presbyterian Hospital Alumni—Otolaryngology
Morrison Hotel
Walnut Room
Shirley Harold Baron, M.D., Secretary
516 Sutter St.
San Francisco 2, Calif.

Washington University Alumni — Ophthalmology
Bismarck Hotel
David M. Freeman, M.D., Secretary
Washington University
St. Louis 10, Mo.

Washington University Alumni — Otolaryngology
(Cocktails only)
Stevens Hotel
Lower Tower
L. W. Dean, Jr., M.D., Secretary
628 Beaumont Medical Bldg.
St. Louis 8, Mo.

Wilmer Institute Alumni—Johns Hopkins Hospital
Palmer House
Private Dining Rooms Nos. 15 and 16
Robert Day, M.D., Secretary
Johns Hopkins Hospital
Baltimore 5, Md.

Wisconsin University Alumni
Drake Hotel
Camellia House
Charles M. Polan, M.D., Secretary
1042 Sixth Ave.
Huntington 1, W. Va.

PROGRAM IN BRIEF

1950 Meeting — Chicago

SATURDAY, OCTOBER 7, 1950

9:00 a.m.—Council Meeting — Private Dining Room No. 4

SUNDAY, OCTOBER 8, 1950

9:00 a.m.—Registration — Foyer to Grand Ball Room

9:00 a.m.—Ladies' Registration — Private Dining Rooms Nos. 15 and 16

9:00 a.m.—Official Opening of Exhibits: Scientific — Red Lacquer Room
Technical—Exhibition Hall and Red Lacquer Room

10:00 a.m.—Meeting of Joint Committee on Industrial Ophthalmology—Dr. Kuhn's Suite

10:00 a.m.—Meeting of American Committee on Optics and Visual Physiology—Private Dining Room No. 1

12:30 p.m.—Home Study Courses Faculty Luncheon—Private Dining Room No. 9

4:30 p.m.—President's Reception—Grand Ball Room

8:00 p.m.—Special Scientific Program: Committee on Conservation of Hearing and Otosclerosis Study Group Division—Grand Ball Room

MONDAY, OCTOBER 9, 1950

9:00 a.m.—Joint Scientific Session—Grand Ball Room

2:00 p.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—ENT Instruction Courses—Seventh Floor

3:15 p.m.—ENT Instruction Courses—Seventh Floor

4:30 p.m.—ENT Instruction Courses—Seventh Floor

6:30 p.m.—Alumni Dinners

9:00 p.m.—Smoker—Grand Ball Room

TUESDAY, OCTOBER 10, 1950

9:00 a.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—EYE Instruction Courses—Seventh Floor

10:15 a.m.—EYE Instruction Courses—Seventh Floor

11:30 a.m.—EYE Instruction Courses—Seventh Floor

12:45 p.m.—Technical Exhibitors Luncheon—Private Dining Room No. 9

2:00 p.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—ENT Instruction Courses—Seventh Floor

3:15 p.m.—ENT Instruction Courses—Seventh Floor

4:30 p.m.—ENT Instruction Courses—Seventh Floor

5:30 p.m.—Dinner Meeting of American Association of Eye, Ear, Nose and Throat Society Secretaries — Private Dining Room No. 18

8:00 p.m.—Special Scientific Program: Industrial Ophthalmology—Private Dining Room No. 14

8:00 p.m.—Special Scientific Program: American Orthoptic Council and American Association of Orthoptic Technicians—Grand Ball Room

WEDNESDAY, OCTOBER 11, 1950

9:00 a.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—EYE Instruction Courses—Seventh Floor

10:15 a.m.—EYE Instruction Courses—Seventh Floor

11:30 a.m.—EYE Instruction Courses—Seventh Floor

12:45 p.m.—Council Meeting Luncheon—Private Dining Room No. 5

2:00 p.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—ENT Instruction Courses—Seventh Floor

3:15 p.m.—ENT Instruction Courses—Seventh Floor

4:30 p.m.—ENT Instruction Courses—Seventh Floor

7:00 p.m.—Annual Dinner—Grand Ball Room

THURSDAY, OCTOBER 12, 1950

9:00 a.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—EYE Instruction Courses—Seventh Floor

10:15 a.m.—EYE Instruction Courses—Seventh Floor

11:30 a.m.—EYE Instruction Courses—Seventh Floor

12:45 p.m.—Faculty Luncheon—Private Dining Room No. 14

2:00 p.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—ENT Instruction Courses—Seventh Floor

3:15 p.m.—ENT Instruction Courses—Seventh Floor

4:30 p.m.—ENT Instruction Courses—Seventh Floor

5:30 p.m.—Business Meeting—Crystal Room

8:00 p.m.—Special Scientific Program: American Society of Ophthalmologic and Otolaryngologic Allergy—Grand Ball Room

FRIDAY, OCTOBER 13, 1950

9:00 a.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—EYE Instruction Courses—Seventh Floor

10:15 a.m.—EYE Instruction Courses—Seventh Floor

11:30 a.m.—EYE Instruction Courses—Seventh Floor

LOCATION OF ROOMS
SCHEDULED FOR ACADEMY ACTIVITIES

THIRD FLOOR	FOURTH FLOOR	CLUB FLOOR
P.D.R. Nos. 1 through 9	Exhibit Hall	P.D.R. Nos. 14 through 18
P.D.R. No. 11	Red Lacquer Room	
Crystal Room	Foyer to Grand Ball Room	
	Grand Ball Room	
	Executive Office	
	Public Relations Office	

Palmer House
Chicago

P.D.R.—Private Dining Room

Club Floor may be reached by stairway from Fourth Floor

SCIENTIFIC SECTIONS

JOINT SESSION

Monday, October 9, 1950
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
J. MACKENZIE BROWN, M.D.
President

Secretary:
ALGERNON B. REESE, M.D.

Call to Order.....The President

Announcements
John R. Lindsay, M.D.
General Chairman

Introduction of Guest of Honor
John D. Kernan, M.D.
New York, N. Y.

Introduction of Special Guests

Address by the President
A Half Century of Otolaryngology

SCIENTIFIC PROGRAM

The Collagen Diseases
Howard F. Polley, M.D.
Rochester, Minn.
BY INVITATION

The Viruses
Thomas Francis, Jr., M.D.
Ann Arbor, Mich.
BY INVITATION

The Antibiotics
Perrin H. Long, M.D.
Baltimore, Md.
BY INVITATION

AUTHORS OF PAPERS

All slides or films must be in the hands of the projection operator before the opening of each session.

Please be prepared to submit papers to the recorder immediately following presentation.

SECTION ON OPHTHALMOLOGY

At the beginning of each scientific session a clinicopathologic case report will be given correlating the clinical with the pathologic findings.

Monday, October 9, 1950
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
EDWIN B. DUNPHY, M.D.
First Vice-President

Secretary:
ALGERNON B. REESE, M.D.

CONGENITAL CYST OF THE OPTIC NERVE WITH ENCEPHALOCELE
(A Clinicopathologic Case Report)
Georgiana Dvorak-Theobald, M.D.
Oak Park, Ill.

Male, age 18 months; left eye deviated down and out since birth. Moderate proptosis; apparently blind.

This specimen presents the very unusual picture of a congenital cyst interposed between the optic nerve and its arachnoid sheath on the nasal side and extending into the posterior part of the globe through a defect in the sclera. Within the cyst are strands of tissue composed of a stroma in which are many large pale staining cells with prominent nucleoli, ganglion cells and smaller deeply staining cells. The strand adjacent to the nerve is confluent with it. This tissue has the appearance of normal brain tissue. The posterior end of the cyst is cut off at the neurectomy, and it seems likely it may extend backward along the optic nerve to become continuous with the brain, thus forming a true encephalocele. Roentgenograms of the skull and optic foramen were negative.

2:10 p.m.
SYMPOSIUM: CORNEAL DISEASES

Chairman:
Frederick C. Cordes, M.D.
San Francisco, Calif.

Normal Variations of the Cornea
David Cogan, M.D.
Belmont, Mass.

Inflammations
James H. Allen, M.D.
New Orleans, La.

Degeneration and Dystrophies
F. Phinizy Calhoun, Jr., M.D.
Atlanta, Ga.

Classification of Diseases
Edwin B. Dunphy, M.D.
Boston, Mass.

Treatment
Kenneth C. Swan, M.D.
Portland, Ore.

Summary
Frederick C. Cordes, M.D.

Questions and Answers

Written questions from the floor can be submitted and will be directed to the participant dealing with that phase of the subject.

SECTION ON OTOLARYNGOLOGY

Tuesday, October 10, 1950
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
KENNETH L. CRAFT, M.D.
Second Vice-President

Secretary:
JAMES H. MAXWELL, M.D.

THE PATHOLOGY OF CARCINOMA OF THE LARYNX BASED ON SERIAL SECTIONS

(Address by the Guest of Honor)

John D. Kernan, M.D.
New York, N. Y.
BY INVITATION

9:30 a.m.

DEAFNESS FOLLOWING BLOWS TO THE HEAD: A CLINICAL AND EXPERIMENTAL STUDY

Harold F. Schuknecht, M.D.
Chicago, Ill.
BY INVITATION

Discussers:

E. Martin Freund, M.D.
Albany, N. Y.

Stacy Guild, Ph.D.
Baltimore, Md.
BY INVITATION

The purpose of this study is to demonstrate the pathology underlying the inner ear (perception) type of deafness which may follow a blow to the head. This condition is often termed inner ear concussion or commotio labyrinthi.

Ten cats were motor-conditioned to pure tone auditory stimuli, and threshold audiograms were made. The animals were then subjected to head blows. Threshold audiograms were again made and at varying periods the animals were sacrificed. The pathologic changes within the inner ear and brain were determined and correlated with the hearing test. The cochleas were graphically reconstructed to show the location of the pathologic changes.

The hearing losses were most severe in the region of the 4096 frequency but in the more severe cases involved the entire audible frequency range—which also occurs in humans.

The injury was primarily to the organ of Corti, followed by degeneration of the nerve fibers and ganglion cells. The damage was most severe in the upper part of the basal coil.

Hemorrhages were present within the inner ears of five animals. Over a period of many weeks this blood caused no apparent reaction within the labyrinth and appeared to have no significance in the cause of deafness in these animals.

The findings indicate that a blow to the head creates a shock pulse which injures the inner ear. The cochlear changes resemble those which are seen following an air-borne shock pulse, such as occurs in blast injury.

10:10 a.m.

THE INNER EAR

(A Motion Picture)

H. G. Kobrak, M.D., Ph.D.
and
J. E. Hind
BY INVITATION
Chicago, Ill.

In the past, nearly all research on the inner ear has been done by histologic methods. This film is based on the assumption that microscopic technics cannot solve important problems of the inner ear and should be amended by macroscopic cinematographic recordings. The technics used in this film are: (1) optical

enlargement of the structures; (2) time magnification; (3) artificial transparency of the membranes in the inner ear; and (4) demonstration of fluid movements by suspended particles. It was possible to record acoustic oscillations in the cochlea and to observe some phenomena in the stratoreceptor.

10:30 a. m.

HEMATOLOGIC ASPECTS OF OTOLARYNGOLOGY

Frank H. Bethell, M. D.
Ann Arbor, Mich.
BY INVITATION

Discussers:
Willis M. Fowler, M. D.
Iowa City, Iowa
BY INVITATION
Russell A. Sage, M. D.
Indianapolis, Ind.

The specialized fields of the otolaryngologist and the hematologist bring them together at a number of points. Such common interests involve a wide range of disorders, all of which are clinically important and in all of which treatment and prognosis depend upon accurate diagnosis. These conditions include certain anemias, especially the chronic iron deficiency anemia associated with dysphagia, known as the Plummer-Vinson syndrome; hemorrhagic disorders such as the purpuras, hemophilia and hereditary hemorrhagic telangiectasia; inflammatory conditions, such as agranulocytic angina and infectious mononucleosis; and neoplastic diseases, especially leukemia, Hodgkin's disease, and lymphosarcoma. Patients with any of these disorders frequently present complaints which bring them first to the attention of the otolaryngologist. Their adequate management requires consultation and continued collaboration with the internist or clinical pathologist who is especially interested in hematology.

11:10 a. m.

A HISTORICAL MOTION PICTURE
Presented through the courtesy of
Julius Lempert, M. D.
New York, N. Y.

A motion picture of historical interest demonstrating a submucous resection of the nasal septum.

11:30 a. m.

DIFFUSE EXTERNAL OTITIS: ITS PATHOLOGY AND TREATMENT

Ben H. Senturia, M. D.
St. Louis, Mo.

Discussers:
Francis E. Senear, M. D.
Chicago, Ill.
BY INVITATION
W. D. Gill, M. D.
San Antonio, Texas

Diffuse external otitis is a disease entity which is known throughout the tropical and subtropical world as "swimming ear," "tropical otitis," "hot weather ear," etc. In recent years in the United States it has been referred to frequently as "fungus ear," and much emphasis has been given to the finding and treatment of fungi in the ear canal.

As a result of recent bacteriologic and mycologic studies of this condition, the "fungus" concept of etiology has been discarded and more consideration has been given to the finding of gram-negative bacilli. As a consequence, the search for a satisfactory form of therapy has turned away from the use of fungicides toward an attempt to discover more adequate bactericidal agents.

However, it is the writer's belief that diffuse external otitis is not, fundamentally, a disease caused by micro-organisms and that it is not caused by swimming. It is felt that the micro-organisms are associated or secondary invaders, while participation in the sport is a coincidental environmental factor which contributes to the pathologic state of the skin of the external auditory canal.

Physiologic, pathologic and clinical evidence will be presented to support the thesis that there are characteristic alterations of the epidermis, corium and skin appendages which are directly responsible for the clinical entity referred to as diffuse external otitis.

For the past two years treatment of all our cases of diffuse external otitis has been predicated upon this concept of pathogenesis, with gratifying results. The various methods of treatment will be discussed.

SECTION ON OPHTHALMOLOGY

Tuesday, October 10, 1950
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
JAMES MASON BAIRD, M.D.
Third Vice-President
Secretary:
ALGERNON B. REESE, M.D.

BILATERAL CYSTIC MASSIVE SEPARATION OF THE RETINA IN A JUVENILE

(A Clinicopathologic Case Report)

Parker Heath, M.D.
and
Joseph Ginsberg, M.D.
BY INVITATION
Boston, Mass.

A report is given of spontaneous bilateral separation of the retina, apparently primarily cystic in character, occurring in a boy 12 years old. The patient had a chondrodysplasia manifested by progressive inability to walk and to sit erect and associated with painless swellings in his joints, without fever, all beginning about the age of 1 year. Unlike secondary and degenerative cystic changes in the retina, this patient maintained fairly good vision in spite of the extensive, semitransparent cysts visible in both eyes with the ophthalmoscope. One eye was removed because of blindness and discomfort incident to secondary glaucoma. The vision in the other eye was maintained somewhat following surface diathermy coagulation. The specimen to be illustrated showed bilateral cystic involvement and secondary separation of the retina. The patient's ocular pathologic condition is classified as a massive cystic separation of the retina, possibly due to pathologic vascular permeability.

2:10 p.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

2:20 p.m.

EMBRYOLOGY OF THE EYE

(A Motion Picture)

Sponsored by the American Academy of Ophthalmology and Otolaryngology

3:30 p.m.

THE OCULAR FINDINGS OF INTRACRANIAL TUMOR

(A Study of 358 Cases)

Manousos Angel Petrohelos, M.D.
BY INVITATION
and
John Woodworth Henderson, M.D.
Ann Arbor, Mich.

Discusser:
Donald J. Lyle, M.D.
Cincinnati, Ohio

The diagnosis and successful localization of intracranial neoplasms should ideally be the result of close co-operation between the neurosurgeon, neurologist and the ophthalmologist. Such a relationship exists at the University Hospital of the University of Michigan, where the neurosurgery and neurology departments routinely refer the great majority of their suspected cases of intracranial pathology for ophthalmologic consultation.

A group of 358 cases of verified intracranial tumor was selected in order to include all intracranial sites of neoplasm. The relative incidence of ocular findings was classified according to each anatomic location of tumor growth. These findings included papilledema, optic atrophy, visual field changes, pupillary abnormalities, defects of conjugate movement, nystagmus, and extraocular muscle palsies.

Slides will be presented showing the incidence of each finding.

3:55 p.m.

FUNDUS CHANGES BEFORE AND AFTER THE RICE DIET

(A Kodachrome Demonstration)

Wm. Banks Anderson, M.D.
Durham, N. C.

Discusser:
Robert W. Hollenhorst, M.D.
Rochester, Minn.

Hypertensive cardiovascular renal disease is of interest to ophthalmologists, first, because many patients seeking relief for headaches or blurred vision are found to be suffering from this disease and, second, because such patients are frequently referred to ophthalmologists for evaluation of the degree of retinal damage, variations in retinopathy associated with hypertensive cardiovascular renal disease being utilized to gauge the effectiveness of therapy and to afford a prognostic index. In a study of 1000

cases of hypertensive cardiovascular renal disease treated with the rice diet, a diet which contains less sodium, less chloride, less protein and less cholesterol than any other diet, 310 were found at the beginning of the treatment to have hemorrhages, exudates, or papilledema, frequently in combination. In 183 of the 310 patients, fundus photographs taken both before and after one to thirty-one months of the rice diet are available for comparison. Studies of these photographs indicate that the retinopathy of hypertensive cardiovascular renal disease, previously considered of such serious prognostic import, is within limits reversible by the rice diet, that such regression is associated with improvement of the patient's well-being and that, contrary to previous opinion, a retinopathy which recedes under proper dietary regimen may recur when such restrictions are relaxed.

4:15 p.m.

PATHOLOGY OF THE EYE IN OLD AGE

A. L. Kornzweig, M.D.
New York, N. Y.

Discusser:

Benjamin Rones, M.D.
Washington, D. C.

This report is based upon the postmortem examination of 120 eyes of inmates of the Home for Aged and Infirm Hebrews of New York City. The ages, at the time of death, were mostly between 70 and 90 years, a few over 60 and several over 90 years of age. These patients had been followed in the eye clinic for several years, and the condition of the functioning eye was noted. Most of the eyes were relatively normal for this age group, but a few of the patients had specific eye diseases such as glaucoma, detachment of the retina, high myopia, status after cataract extraction, retinal vein and artery thromboses, and diabetic retinopathy. Seventy-eight per cent, however, had vision that was 20/70 or better. The usual pathologic changes associated with old age were recorded and classified according to the frequency and degree of occurrence. The unusual pathologic findings were also listed, even if they occurred but once.

In this way a clinical and pathologic correlation was possible in most of the eyes obtained after death. This report, however, deals chiefly with the pathologic findings.

4:45 p.m.

CORNEAL CONTACT LENSES

(A Motion Picture)

Louis J. Girard, M.D.
New York, N. Y.
BY INVITATION

The normal metabolism of the cornea requires adequate circulation of blood at the limbus and bathing and circulation of tears. Capping of the cornea with a watertight seal, such as is produced by the conventional contact lens, interferes with these physiologic processes, and blurring, halos, and discomfort usually result within two to three hours.

In October 1948 our attention was drawn to the Tuohy corneal lens, a new type contact lens which requires no accessory fluid and which consists of a corneal part only. This lens appears to be a definite advance in type and fitting of contact lenses. A description of the lens and the fitting procedure is presented.

SECTION ON OTOLARYNGOLOGY

Wednesday, October 11, 1950

9:00 a.m.

GRAND BALL ROOM

Presiding Officer:

J. MACKENZIE BROWN, M.D.
President

Secretary:

JAMES H. MAXWELL, M.D.

THE MANAGEMENT OF DEAFNESS

(Wherry Memorial Lecture)

Kenneth M. Day, M.D.
Pittsburgh, Pa.

9:30 a.m.

SYMPOSIUM: MALIGNANT TUMORS

Moderator:

Gordon B. New, M.D.
Rochester, Minn.

Malignant Neoplasms of the Nose, Paranasal Sinuses, and Nasopharynx: Evaluation of Surgical Treatment

LeRoy A. Schall, M.D.
Boston, Mass.

An analysis of an extensive experience with neoplastic disease involving the nose, nasal accessory sinuses, or both reveals that surgery followed by adequate irradiation gave the patient a twice greater possibility of a five year survival than any other form of treatment.

The surgery must be extensive, thorough and, when possible, through normal tissue. The exposure must be adequate. The region involved should determine the approach. When the neoplastic disease is limited to the nasal cavity, the approach may be by way of a lateral rhinotomy. Differing from some operators, the author believes that the lower part of this incision should not extend into the anterior nares. The vestibule of the nose should be respected, and good exposure can be obtained by extending the incision under the vestibule but not into it.

Should the disease prove to be more extensive than anticipated, this incision may be extended upward to expose the frontal sinus, down through the upper lip, and, with a buccal incision to the last molar tooth, the whole cheek can be reflected to expose the superior maxilla.

Should the orbit be the part of greatest involvement, with exophthalmos, destruction of the orbital contents and fixation, then the approach should be by way of the orbit by an exenteration of the orbital contents. This approach affords access to the frontal sinus from below, to the ethmoidal labyrinth, sphenoid sinus and nasal cavity from inner wall of the orbit, and to the maxillary sinus from above by removal of the floor of the orbit.

When the floor of the maxillary sinus or the floor of the nose is involved either from a lesion primary in the antrum or by extension from a primary lesion of the aveolus or hard palate, then the approach may be from below by resection of the palate, with or without splitting the lip, and a modified lateral rhinotomy depending upon the extent of the lesion.

With the exception of the benign new growths, surgery has no place in the treatment of neoplastic disease of the nasopharynx. Radiation either by radium or by external irradiation has proved so superior to surgery that in the modern treatment of nasopharyngeal malignancy surgery should be discarded.

Malignant Neoplasms of the Nose, Paranasal Sinuses, and Nasopharynx: Evaluation of Radiation Therapy

Maurice Lenz, M.D.
New York, N. Y.

BY INVITATION

Malignant neoplasms of the nose, paranasal sinuses and nasopharynx are best treated by a team consisting of an otolaryngologist and radiotherapist. Successful radiotherapy requires the inclusion in the irradiated field of the primary growth and all its extensions and metastases and the administration of a sufficient radiation dose to destroy the cancer. Familiarity with the local anatomy, the usual routes of extension and the clinical characteristics of the individual cancer are indispensable for correct localization of radiotherapy. Cancericidal radiation may be administered most readily if the cancer is small, easily accessible, has a tendency to grow superficially and to exuberate, has not metastasized, and is radiosensitive. It becomes much more difficult if the growth is extensive, less accessible, tends to infiltrate, especially into bone, has metastasized to distant parts, and is less radioresponsive. Unless they have extended beyond the radiated area, lymphosarcomas and lymphoepitheliomas are more radiocurable than well-differentiated epitheliomas, cylindromas or other adenocarcinomas.

The inaccessibility of nasopharyngeal growths to complete excision and the frequency with which these neoplasms prove radiosensitive make nasopharyngeal cancers especially suited for radiotherapy. From 1926 to 1945, over 90 patients were given radiotherapy for malignant neoplasm of the nasopharynx at the Manhattan Eye, Ear and Throat Hospital, Montefiore and Presbyterian hospitals, New York. About one-third of these patients survived five years, and roughly one-quarter, ten years after radiotherapy. Those which did best were lymphosarcomas and lymphoepitheliomas limited to the head and neck; most failures in this group being due to distant metastases. Those patients with cylindromas and well-differentiated epitheliomas responded least, most dying from local persistence of the disease. Bone invasion was frequent among epitheliomas and exceptional in lymphosarcomas. Prognostically, skull invasion was serious except in

lymphoepitheliomas, in which 4 of 6 patients showing this involvement remained well for five years. Involvement of cranial nerves was noted in one-third of the cases and was especially frequent among those classified as slightly differentiated epitheliomas. The cancers arising near the fossa of Rosenmuller had a poorer prognosis than those originating from the region of the pharyngeal tonsil. The latter location was the primary site of most lymphosarcomas.

Cancers of the maxillary antrum readily invade the bony walls and often are not radioresponsive; most of these growths are treated best by a combination of surgery and radiotherapy. Of 12 malignant neoplasms of the maxillary antrum receiving radiotherapy at the Manhattan Eye, Ear and Throat Hospital from 1943 to 1945, 2 are now symptom-free. Of 18 similar growths treated by irradiation at the Presbyterian Hospital from 1931 to 1945, 3 are living and 2 of these are symptom-free. One of these 4 symptom-free cases was treated by surgery followed by radiotherapy, and 3 received only roentgen therapy. In contrast to these poor results in cancer of the antrum treated chiefly by radiation in a very advanced stage, the reports from the Radiumhemmet in Stockholm state that their results show that more than 40 per cent of their patients with this disease remain well five years or longer following treatment combining excision, electrocoagulation, radium and roentgen therapy.

The best results in the treatment of malignant neoplasms of the nose, paranasal sinuses and nasopharynx result from collaboration of an otolaryngologist and radiotherapist over a period of years.

Malignant Neoplasms of the Tongue, Tonsil, and Hypopharynx: Evaluation of Surgical Treatment

Henry B. Orton, M.D.
Newark, N. J.

Malignant Neoplasms of the Tongue, Tonsil, and Hypopharynx: Evaluation of Radiation Therapy

Isadore Lampe, M.D.
Ann Arbor, Mich.

BY INVITATION

Malignant lesions of the tongue, tonsil and hypopharynx (including epiglottis, aryepiglottic folds and postcricoid region) constitute a group of tumors diversified in clinical manifestations, histopathologic characteristics and natural history. The diversification is such that, although such knowledge is essential in the determination of proper treatment, only highlights can be pointed out.

In most of these tumors irradiation has been employed as a therapeutic procedure either alone or in association with surgical procedures. An attempt will be made to present briefly current thought in radiotherapy regarding the lesions in which irradiation is indicated. An estimate of the results that can be expected will be made.

Question and Answer Period

During the symposium, written questions may be presented to the Secretary. These will be given to the Moderator for distribution to participants in the symposium.

11:30 a.m.

ENDAURAL SUBCORTICAL MASTOIDOTYMPANECTOMY FOR THE CURE OF CHRONIC PERSISTENT SUPPURATIVE OTITIS MEDIA

(A Motion Picture)

Julius Lempert, M.D.
New York, N. Y.

SECTION ON OPHTHALMOLOGY

Wednesday, October 11, 1950
2:00 p.m.

GRAND BALL ROOM

Presiding Officer:
EDWIN B. DUNPHY, M.D.
First Vice-President

Secretary:
ALGERNON B. REESE, M.D.

MALIGNANT LYMPHOMA OF THE UVEAL TRACT

(A Clinicopathologic Case Report)

Edmond L. Cooper, M.D.
Detroit, Mich.
and
John L. Riker, M.D.
Alpena, Mich.

BY INVITATION

A 26-year old white man presented symptoms which simulated typical uveal inflammation of the chronic, granulomatous type. The proper diagnosis of malignant lymphoma of the eyeball was made only upon pathologic examination of the enucleated eye. In the early months of the condition the only signs and symptoms were ocular, although later the tumor became more widespread, involving the lymph glands and testicles. The ocular process was probably either metastatic or part of a widely disseminated lymphoid activity. In any case of severe uveitis with nodular iritis and retinal hemorrhages for which no cause is found and which does not respond to treatment, it is important to consider the possibility of malignant lymphoid disease.

2:10 p.m.

OPTIC NERVE SHEATH HEMORRHAGE

(Jackson Memorial Lecture)

Frank B. Walsh, M.D.
Baltimore, Md.

2:45 p.m.

SURGERY OF THE SUPERIOR OBLIQUE

(A Motion Picture)

Walter H. Fink, M.D.
Minneapolis, Minn.

The purpose of the presentation is to describe certain procedures employed in surgery of the superior oblique. The presentation expresses a viewpoint in a field of ophthalmic surgery which is apparently in a transitory phase.

The technics advocated seem most feasible from the anatomic standpoint and most effective from the functional standpoint.

The subject is presented in three phases:

1. Anatomic Considerations
   Various anatomic features are described which determine the type of operative procedure.
2. Diagrammatic Presentation
   The operative technics are described by means of drawings which show the various steps in the procedures.
3. Operative Procedures
   The following operations are shown:
   a. Tenotomy of the superior oblique tendon
   b. Tuck of the superior oblique tendon
   c. Resection of the superior oblique tendon (with and without tenotomy of the superior rectus)

3:10 p.m.

BRUCELLOSIS

Ophthalmology's Stake in Brucellosis

Alan C. Woods, M.D.
Baltimore, Md.

Ocular lesions, keratitis and uveitis, occur in about 10 per cent of guinea pigs and rabbits with systemic Brucella infections. Clinical ocular lesions from brucellosis were first recognized in 1924. Since then our knowledge in the ocular lesions of brucellosis has been considerably amplified. The ocular symptomatology and pathology are briefly reviewed. It is now believed that Brucella infection accounts for 5 to 10 per cent of endogenous uveitis.

Human Brucellosis (Undulant Fever)

Wesley W. Spink, M.D.
Minneapolis, Minn.

BY INVITATION

Brucellosis is the most important human disease transmitted from domestic animals to man. Three species of Brucella are the cause of human disease: Br. melitensis, Br. suis, and Br. abortus. The disease is acquired through direct contact with infected material, or through ingestion of contaminated cows' and goats' milk. The illness may be acute and febrile, or very insidious in its onset. The diagnosis can only be established by laboratory aids, such as the agglutination reaction and blood cultures. Very encouraging results in treatment have been obtained with the antibiotics, particularly aureomycin.

Questions and Answers

4:00 p.m.

ADHERENCE SYNDROME: PSEUDOPARALYSIS OF THE LATERAL OR SUPERIOR RECTI

(A Motion Picture)

Lorand V. Johnson, M.D.
Cleveland, Ohio

Fink has described the normal attachments between the sheaths of the recti and obliques, Berke has reported on the obliques, and Scobee has emphasized the

importance of check ligaments, "footplates," strabismus fixus, and their surgical significance.

The present discussion treats with still another anomaly of the muscle sheaths, seldom present but of important surgical consideration. This anomaly is of firm adherence between the sheaths at two points: (1) between the sheaths of the lateral rectus and the inferior oblique, and (2) between the sheaths of the superior rectus and the superior oblique. This adherence is considered to be a manifestation of faulty development in the loose, physiologic attachments as described by Fink. It occurs quite frequently before the child is 3 years of age, occasionally between the ages of 3 and 5, and seldom after the age of 7 years.

Diagnosis by "fixed-muscle" duction, demonstration of types of adherence, and surgical considerations of loose and firm adherence are illustrated by motion pictures.

4:20 p.m.

NEMATODE ENDOPHTHALMITIS
Helenor Campbell Wilder
Washington, D. C.

Discusser:
John S. McGavic, M.D.
Bryn Mawr, Pa.

A recent review of cases of endophthalmitis in the Registry of Ophthalmic Pathology at the Armed Forces Institute of Pathology disclosed a group in which nematode larvae in the posterior segment of the globe were demonstrated by serial sectioning. Almost all the patients were children under 10 years of age who lived in southeastern United States, although they were not limited to this region. The usual clinical diagnosis was retinoblastoma. The inflammatory reaction was composed largely of eosinophilic abscesses, epithelioid cells with occasional giant cells, lymphocytes, and numerous plasma cells, many of which were multinucleated. An inflammatory membrane generally occupied the funnel of the detached retina. Hemorrhages were a frequent complication. Although the occurrence of parasites in the eye in nematodiasis is well known, it is believed that they play an important and hitherto unrecognized role in the production of pseudoglioma and possibly Coats's disease.

SECTION ON OTOLARYNGOLOGY

Thursday, October 12, 1950
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
KENNETH L. CRAFT, M.D.
Second Vice-President

Secretary:
JAMES H. MAXWELL, M.D.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:20 a.m.

PROBLEMS OF THE SEPTUM IN RHINOPLASTIC SURGERY
Oscar J. Becker, M.D.
Chicago, Ill.

Discussers:
Thomas C. Galloway, M.D.
Evanston, Ill.
John M. Converse, M.D.
New York, N. Y.

The management of an anterior dislocation and deviation of the septum, when it is combined with the reconstruction of an external nasal deformity, is dependent upon the pathologic condition present. No single method is completely applicable in every type of case. The choice of operative procedure is dependent, furthermore, upon whether or not a combined septum and plastic operation is contemplated, since the problem is distinctly different when the septal operation is done alone.

It has been my experience in correcting a twisted or hypertrophied nose, combined in a one-stage operation with a dislocated, deviated septum, that the "Metzenbaum type" of operation is a satisfactory method in the majority of such cases. However, several shortcomings and objectionable features of the "Metzenbaum type" technic have led to the development of certain modifications which help overcome some of the difficulties encountered in its application.

Complete removal of the septum, with or without replacement of cartilage, is a procedure to be employed only in cases which cannot be corrected by other means. It is indicated only in selected cases rather than as a routine method in rhinoplastic surgery combined with a submucous resection.

10:00 a.m.

THE SURGICAL TREATMENT OF FACIAL PARALYSIS

(A Motion Picture)

Joseph A. Sullivan, M.D.
Toronto, Ont., Can.

This picture illustrates in color the endaural approach for the repair of a damaged facial nerve as a result of a mastoid operation. The examination of the nerve under the dissecting microscope reveals the extent of the injury and a grossly swollen nerve. The careful incision of the neurilemmal connective tissue sheath is brought out in detail. The necessity of the conversion of a previous simple mastoidectomy into the radical is shown in order to provide adequate exposure of the nerve above the site of injury. The graft was prepared in the leg, but the lesion as evidenced under the microscope obviated the necessity of its use. The removal of the stylomastoid foramen to the division of the nerve at the pes anserinus is clearly seen even through the endaural approach. The appearance of the face is shown six months after operation.

10:20 a.m.

AN EVALUATION OF COCAINE ANESTHESIA

A. C. Furstenberg, M.D.
and
Lauren A. Woods, M.D., Ph.D.
BY INVITATION
John E. Magielski, M.D.
BY INVITATION
Ann Arbor, Mich.

Discussers:

H. L. Williams, M.D.
Rochester, Minn.
A. L. Tatum, M.D., Ph.D.
Madison, Wis.
BY INVITATION

11:00 a.m.

TOTAL RECONSTRUCTION OF THE EAR BY PREFABRICATED FRAMEWORK OF REFRIGERATED BONE AND CARTILAGE

(A Motion Picture)

John M. Converse, M.D.
New York, N. Y.

This motion picture demonstrates a new technic being employed in the Plastic Surgery Clinic of the Manhattan Eye, Ear and Throat Hospital. Preservation of bone and cartilage by refrigeration in a bone bank has permitted leisurely prefabrication of an adequate auricular framework. Using the technic outlined, partial and total ear reconstruction has been achieved in two or three operations over a treatment period of from three to six months.

11:20 a.m.

THE CONCEPT OF ALLERGY AS AUTONOMIC DYSFUNCTION: AN IMPROVED WORKING HYPOTHESIS FOR CLINICAL ALLERGY

H. L. Williams, M.D.
Rochester, Minn.

Discussers:

Rea Ashley, M.D.
San Francisco, Calif.
John M. Sheldon, M.D.
Ann Arbor, Mich.
BY INVITATION

Von Pirquet's hypothesis of allergy, formed from his own work and that of Ehrlich and Arthus and Breton, was evolved for the purpose of explaining certain observed clinical phenomena. When it was found that clinical allergy in many cases did not meet the criterion of demonstrable circulating antibodies established by the hypothesis, instead of admitting that the fundamental assumption was incorrect and either abandoning or correcting the hypothesis, immunologists and allergists decided to restrict the term "allergy" to those instances of the clinical phenomenon of allergy in which an antigen-antibody type of reaction could be demonstrated. It would appear, however, that the term "allergy" was originally coined to describe the clinical phenomenon and not the hypothesis. When, however, the majority of allergies failed to meet the criterion of circulating antibodies established by the hypothesis, instead of entertaining the possibility that there might be some defect in this fundamental assumption, many immunologists insisted that only that small portion of those indistinguishable clinical phenomena in which circulating antibodies could be detected could rightfully be called "allergy." Some allergists, particularly Duke, were not satisfied with this viewpoint.

It is felt that by research of the literature on the body's method of resistance to changes in the external and internal environment and by considering the de-

velopment of a resistance mechanism through phylogeny, a broader and more physiologic concept, interdigitating better with the observed facts, could be suggested.

It would seem reasonable, therefore, to speak of three related but not identical types of allergy: (1) physical allergy (reflex type); (2) bacterial or tissue allergy, and (3) humoral allergy.

In physical allergy (reflex type), no antigen-antibody type of reaction occurs. Vasospasm and anoxia result in vascular injury, increased capillary permeability, release of histamine and the production of the typical allergic wheal. It is phylogenetically the most primitive type of allergy and can be considered an overaction of the "alarm reaction" described by Selye.

In bacterial or tissue allergy the primitive vascular reaction is retained but cell injury also may be produced by an antigen-antibody type of mechanism retained in the tissue cells. There are no freely circulating antibodies in the blood stream. The normal physiologic prototype of which this type of allergy could be considered the hyperfunction is granulomatous inflammation.

In humoral allergy, retaining the primitive reaction, an antigen-antibody reaction in the cells is also retained, but in addition circulating antibodies, probably from the lymphocyte, are present in the blood serum. Suppurative inflammation could be considered the normal prototype of which humoral allergy is a hyperfunction.

There is nothing in this new concept of allergy to suggest that these three types of allergy are mutually exclusive.

The proposed phylogenetic concept presents allergy as a gradual growth in the animal organism of an increasingly more elaborate defense mechanism, new developments being added to the primitive mechanism rather than replacing it; all types of allergy, therefore, consist of hyperfunction of this stereotyped defense mechanism.

SECTION ON OPHTHALMOLOGY

Thursday, October 12, 1950
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
JAMES MASON BAIRD, M.D.
Third Vice-President
Secretary:
ALGERNON B. REESE, M.D.

BILATERAL UVEITIS AND KERATITIS ACCOMPANYING PERIARTERITIS NODOSA

(A Clinicopathologic Case Report)
Raymond G. Ingalls, M.D.
New York, N. Y.

The eye manifestations followed the pattern of any severe uveitis.

The diagnosis of periarteritis nodosa was established by autopsy findings.

2:05 p.m.
PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

2:15 p.m.
THE COLLAGEN DISEASES

The Pathology of Collagen Disease
Leonard Christensen, M.D.
Portland, Ore.
BY INVITATION

Of the three basic elements of connective tissue, i.e., collagen, reticular and elastic fibers, collagen has assumed the greatest importance. Pathologic alteration of this tissue is the apparent anlage of several previously unrelated diseases, such as lupus erythematosus, periarteritis nodosa, polyarthritis and scleromalacia perforans.

These diseases are collectively termed as diseases of the collagen system and the basic lesion is a fibrinoid degeneration of the connective tissue.

Fibrinoid degeneration will be discussed and illustrations of its pathology as it occurs in the eye and other tissues will be presented.

2:30 p.m.
Clinical Observations on Cortisone Therapy in Ophthalmic Disease
John W. Henderson, M.D.
Robert W. Hollenhorst, M.D.
Rochester, Minn.

The discovery and clinical applications of cortisone, a hormone of the adrenal cortex, prior to its use in ophthalmology will be briefly reviewed. The effects of cortisone on ophthalmic diseases such as keratitis, uveitis, scleritis, chorioretinitis,

and vernal conjunctivitis will be discussed and illustrated. Some of the ophthalmic aspects of the so-called collagen diseases will be mentioned, and the effect of cortisone on these diseases will be considered.

2:55 p.m.

Clinical Experiences with ACTH and Cortisone in Ocular Diseases

John M. McLean, M.D.
New York, N. Y.

Clinical results in over 75 cases and a wide variety of ocular diseases treated parenterally with ACTH, cortisone, or both are reported. Complications and dangers, both systemic and ocular, are outlined. The results of local treatment with cortisone in about a hundred cases, both alone and in conjunction with parenteral therapy, are outlined. Suggestions as to mechanism of action and clinical applicability are offered.

3:15 p.m.

Questions and Answers

3:25 p.m.

THE DYNAMICS OF PHAKOERISIS
(A Motion Picture)

David O. Harrington, M.D.
San Francisco, Calif.

The increasing popularity of the suction method of intracapsular cataract extraction makes it important to understand the dynamics of the procedure.

The modern erisophake with its limited and self-controlled vacuum has eliminated many of the hazards of the method and made it a safe procedure in average hands.

Motion picture analysis of erisophake extraction of lenses from living human eyes and cadaver eyes clearly demonstrates the dynamics of the method, especially when filmed under high magnification and in slow motion. By this method the following facts are demonstrated:

1. Application of suction to the anterior lens capsule draws the capsule forward into the suction cup.
2. The lines of capsular stress are transmitted to the equatorial region which is pulled forward, thus tightening the zonule and drawing it tangential to the equator so that it is easily peeled from the capsule.
3. Little or no external pressure is required to rupture the zonule, and capsular rupture is rare.
4. The procedure is applicable to all types of cataract.

A new erisophake is demonstrated.

3:50 p.m.

PROGRESS IN DETACHMENT SURGERY

Charles L. Schepens, M.D.
Boston, Mass.

BY INVITATION

Discusser:

Peter C. Kronfeld, M.D.
Chicago, Ill.

Three problems are discussed. The first one refers to difficulties resulting from marked changes of the intraocular pressure during operation. Nonperforating diathermy increases the tension and this causes corneal edema. Sudden release of the subretinal fluid may cause macular or papillar hemorrhage. In cases of reoperation, increase of the tension may rupture the globe. Procedures which are used to avoid these complications are discussed.

The second problem concerns pupillary dilatation and maintenance of corneal transparency. Various methods for dilating the pupil are mentioned, and the subconjunctival injection of a mixture containing neo-synephrine, homatropine and procaine is recommended. The steps taken to preserve the corneal transparency during operation are described.

The third problem is related to ophthalmoscopy of detachments. With a direct ophthalmoscope, one can only investigate the posterior two-thirds of the retina. A new type of indirect stereoscopic ophthalmoscope is described, which is an improved version of a model demonstrated in 1946. For examination of the extreme periphery, the sclera is indented with a special instrument called scleral depressor. This method enables one to visualize the entire fundus up to the posterior edge of the ciliary processes. Findings are recorded diagrammatically on a special chart. A short motion picture demonstrates how examination of the extreme periphery is performed, using this new method of ophthalmoscopy.

4:20 p.m.

THE CONTAMINATION OF OPERATIVE WOUNDS WITH COTTON FIBRILS AND TALC

L. R. Duszynski, M.D.
New York, N. Y.
BY INVITATION

Discusser:

Derrick Vail, M.D.
Chicago, Ill.

Attention is called to the occurrence and prevalence of foreign bodies in operative wounds.

Routine sections of globes operated upon for glaucoma and subsequently enucleated were examined with a polaroid microscope. Photomicrographs will be shown demonstrating the presence of foreign bodies and the tissue reaction to them.

The incidence of wound contamination by foreign bodies in glaucoma surgery and the possible significance will be described.

The morphologic and optical properties of the foreign material are those of cotton and/or talc.

SECTION ON OTOLARYNGOLOGY

Friday, October 13, 1950
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
J. MACKENZIE BROWN, M.D.
President

Secretary:
JAMES H. MAXWELL, M.D.

A SURGICAL TECHNIC TO ELIMINATE THE TRACHEOTOMY TUBE AFTER LARYNGECTOMY

Joel J. Pressman, M.D.
Claude S. Mumma, M.D.
Los Angeles, Calif.

Discussers:

Frederick A. Figi, M.D.
Rochester, Minn.
Louis H. Clerf, M.D.
Philadelphia, Pa.

Not infrequently it has been found necessary to repair surgically a tracheal stoma following laryngectomy where cicatricial contraction has narrowed the opening. This cicatricial contraction has occurred not only in patients who have worn a laryngectomy tube constantly but also in those who have dispensed with wearing the tube.

Since it is known that if the cricoid cartilage can be left attached to the trachea and sutured beneath the skin wound, the stoma will remain widely patent, a procedure was developed in which metallic rings were substituted for the excised cricoid and sutured about the trachea subcutaneously at the stoma.

Rings of molded tantalum foil as well as of cast vitallium were used. They were both circular and horseshoe in shape. The rings were placed about the upper tracheal rings externally and sutured to them, as well as to the strap muscles when they were not excised, or to the subcutaneous tissue when they were, by through-and-through, braided tantalum sutures. The sutures were tied externally to the trachea and buried subcutaneously. The operation was then completed, as usual, by drawing the skin into the trachea and securing it with multiple black silk sutures to the inner tracheal wall.

A laryngectomy tube is worn postoperatively until the edema and swelling subside and the silk skin sutures have been removed. In several cases there was a moderate allergic response with formation of granulation tissue which required excision. The rings are well tolerated. The longest time one has been worn with no tissue reaction is twenty-three months.

9:40 a.m.

ENDAURAL INCISIONS
(A Motion Picture)

Walter P. Work, M.D.
San Francisco, Calif.

This colored motion picture depicts the technics for endaural incisions. The contributions of Lempert, Maxwell, Shambaugh, J. F. Tolan, Farrior, Walsh, House, Koerner and others are fully acknowledged.

There are two main divisions in the picture:

1. The technics of incisions for the fenestration and atticomastoidectomy are shown by animated drawings and and by dissection.
2. The technics of incisions for the mastoido-tympanectomy are noted in like manner.

In endaural incisions of this type, an attached pedicle flap is created, swung posteriorly and held by packing at the conclusion of the surgery. Routinely, in all of these incisions approximately 3 mm. of conchal cartilage are removed, ensuring an adequate large well-shaped meatus.

This technic has been used successfully in over one hundred cases without stricture formation.

10:00 a.m.

EXPANSION OF THE LUNGS IN THE NEWBORN: AN EXPERIMENTAL STUDY IN RABBITS

A. C. Hilding, M.D.
and
David Hilding
BY INVITATION
Duluth, Minn.

Discussers:

Paul H. Holinger, M.D.
Chicago, Ill.

James L. Wilson, M.D.
Ann Arbor, Mich.
BY INVITATION

There are many unknown factors involved in the expansion of the lungs in the newborn. There are differences of opinion on the part of investigators as to whether lungs normally expand at all before birth and as to whether expansion is complete shortly after birth. Roentgenograms of a reasonably large number of cases indicate expansion is incomplete immediately after birth. It is a gradual process requiring several days. Our experiments on rabbits indicate expansion is uneven, both as to time and extent. The initial opening of the lungs requires relatively large pressures. The abdominal organs, notably the liver, occupy much of the thoracic space before birth. The ribs are formed about these abdominal organs. The fetus straightens out immediately after birth, thus pulling the abdominal organs caudally and making room for the expanding lungs. The spring of the ribs probably assists in the first breath. Some fetuses apparently expand the lungs well and breathe for a time, then, for some reason, develop more or less complete atelectasis. The lungs from these again sink in water. This atelectasis is probably related to general debility or prematurity. Studies on expansion of the lungs related to prematurity in rabbits are reported.

10:40 a.m.

SURGICAL TREATMENT OF CHRONIC FRONTAL SINUSITIS USING THE SEWALL MUCOPERIOSTEAL FLAP

(A Motion Picture)

Guy L. Boyden, M.D.
Portland, Ore.

The skin incision is a curved one following the inner angle of the orbit, extending outward not farther than the supraorbital notch and downward on the nasal process of the maxillary bone. The floor of the sinus is entirely removed. The elevation of the periosteum exposes the anteroinferior wall of the frontal sinus and the frontal process of the maxillary bone and brings into view the nasal fissure. Just below the nasal fissure, by means of a large burr, the mucoperiosteal flap is uncovered. The different steps will be shown. The cancellous bone of the globella is removed, making a bed for the mucoperiosteal flap. This makes a new opening into the frontal sinus and does away with the use of foreign body drains. The film also shows the technic for tying the anterior ethmoidal artery.

11:00 a.m.

THE RHINOLOGIST'S ROLE IN TEAR-SAC SURGERY

Lester T. Jones, M.D.
Guy L. Boyden, M.D.
Portland, Ore.

Discussers:

A. D. Ruedemann, M.D.
Detroit, Mich.

Louis Daily, M.D.
Houston, Texas

The various methods used to cure epiphora by creating a new ostium in the nose may be summarized by dividing them into two main groups: (1) the methods of Toti-Mosher, Dupuy-Dutemps, and Arruga, and "transplantation," all of which approach the tear sac through an external incision; (2) the West method, which uses an intranasal approach.

From the beginning of the twentieth century until the last few years, well over 80 per cent of our group practiced both ophthalmology and rhinology. In the last few years, not only has the division between these two fields increased, but in our centers of training the residents in rhinology seldom come in contact with either tear-sac pathology or its surgical cure. A survey of resident training cen-

ters for ophthalmology showed that 35 per cent preferred the Toti-Mosher operation, 25 per cent the Dupuy-Dutemps, and 10 per cent the transplantation method. Only 10 per cent referred such cases to the rhinologist for the intranasal procedure. As the ophthalmologist is the first to be consulted for this disease and as the rhinologist is only too often indifferent to the lacrimal apparatus, the drift in America is definitely toward the external approach.

The literature on tear-sac surgery indicates that the percentage of cures by either method is about the same, the chief source of failure being closure of the ostium between the nasal wall of the sac and the nose. This occurs in from 5 to 25 per cent of all cases. This complication, from an anatomic standpoint, lies almost entirely in the field of the rhinologist and such failures can be salvaged most easily by him. A simple office procedure for correction of such failures will be described. Other advantages of the intranasal approach will be listed.

A report on 60 consecutive unselected cases of epiphora, both with and without dacryocystitis, and a description of the technic used with no failures will be given.

11:40 a.m.

CANCER OF THE MOUTH

(A Motion Picture)

John B. Erich, M.D.
Rochester, Minn.

This motion picture on cancer of the mouth shows the methods of therapy which have been found to give the most effective results at the Mayo Clinic. Specifically, malignant lesions of the jaws, tongue, floor of the mouth and cheeks are considered. Special emphasis is placed on the removal of many of these lesions by surgical diathermy (electrocoagulation) and on the selection of treatment which should be in keeping with the activity of the growth, the age and general condition of the patient.

SPECIAL SCIENTIFIC PROGRAMS

OTOSCLEROSIS STUDY GROUP

Division of
Committee on Conservation of Hearing

Private Dining Room No. 14
Saturday, 8:00 p.m.

GEORGE E. SHAMBAUGH, JR., M.D., *President*

JOHN R. LINDSAY, M.D., *Secretary*

Histologic Observations on Fenestrated Human Ears

1. George E. Shambaugh, Jr., M.D., Chicago, Ill., and Hollie E. McHugh, M.D., Montreal, Que., Canada
2. Howard P. House, M.D., Los Angeles, Calif., John R. Lindsay, M.D., Chicago, Ill., Pierre Perret, M.D., Lausanne, Switzerland

Causes for Failure in Fenestration Surgery

Joseph A. Sullivan, M.D., Toronto, Ont., Canada

Theodore E. Walsh, M.D., St. Louis, Mo.

Arthur L. Juers, M.D., Chicago, Ill.

Howard P. House, M.D., Los Angeles, Calif.

Julius Lempert, M.D., New York, N. Y.

COMMITTEE ON CONSERVATION OF HEARING
and
OTOSCLEROSIS STUDY GROUP DIVISION

Grand Ball Room
Sunday, 8:00 p.m.

DEAN M. LIERLE, M.D., *Chairman*

Treatment of Deafness by Mechanical Prosthesis

Heinrich Kobrak, M.D., Chicago, Ill.

Industrial Acoustic Trauma
Douglas Wheeler, Ph.D., Los Angeles, Calif.

New Voices (A Veterans Administration Motion Picture)
Aram Glorig, M.D., Silver Spring, Md.
DISCUSSER:
Scott N. Reger, Ph.D., Iowa City, Iowa

Fenestration Operation for Otosclerosis
(Photography by Paul H. Holinger, M.D.)
George E. Shambaugh, Jr., M.D., Chicago, Ill.

AMERICAN ORTHOPTIC COUNCIL
and
AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

Grand Ball Room
Tuesday, 8:00 p.m.

FRANK D. COSTENBADER, M.D., *Secretary*

Scope and Limitations of Orthoptics in the Nonsurgical Treatment of Ocular Deviations
Hermann Burian, M.D., Boston, Mass.
DISCUSSERS: Julia Lancaster, San Francisco, Calif.
Elizabeth Goggin, Minneapolis, Minn.

The Orthoptist and Hyperphoria
Elizabeth Bennet and Dorothy Thompson, St. Louis, Mo.
DISCUSSERS: Richard G. Scobee, M.D., St. Louis, Mo.
Kenneth C. Swan, M.D., Portland, Ore.

Surgical Treatment of Ocular Deviations: An Important Factor in Orthoptics
Walter B. Lancaster, M.D., Boston, Mass.
DISCUSSERS: Ruth Fisher, Denver, Colo.
Lucia Nugent, Los Angeles, Calif.

INDUSTRIAL OPHTHALMOLOGY
Private Dining Room No. 14
Tuesday, 8:00 p.m.

A. D. RUEDEMANN, M.D., *Chairman*
HEDWIG S. KUHN, M.D., *Secretary*

EYES AND DISASTER

Medical Civil Defense: The Background
Andrew C. Ivy, M.D., Vice President, University of Illinois School of Medicine, Chicago, Ill.

Disaster Strikes: What Happens
Col. Victor A. Byrnes (MC), School of Aviation Medicine, Randolph Field, Texas

Emergency Eye Care in Disaster: The Laymen Who Precede Us
Hedwig S. Kuhn, M.D., Hammond, Ind.

AMERICAN SOCIETY OF OPHTHALMOLOGIC
and
OTOLARYNGOLOGIC ALLERGY

Grand Ball Room
Thursday, 8:00 p.m.

J. W. HAMPSEY, M.D., *Secretary*

History of Allergy in Otolaryngology
French K. Hansel, M.D., St. Louis, Mo.

The Future of Allergy in Otolaryngology and Ophthalmology
M. Murray Peshkin, M.D., New York, N. Y.

Diagnosis of Food Allergy
Theron G. Randolph, M.D., Chicago, Ill.

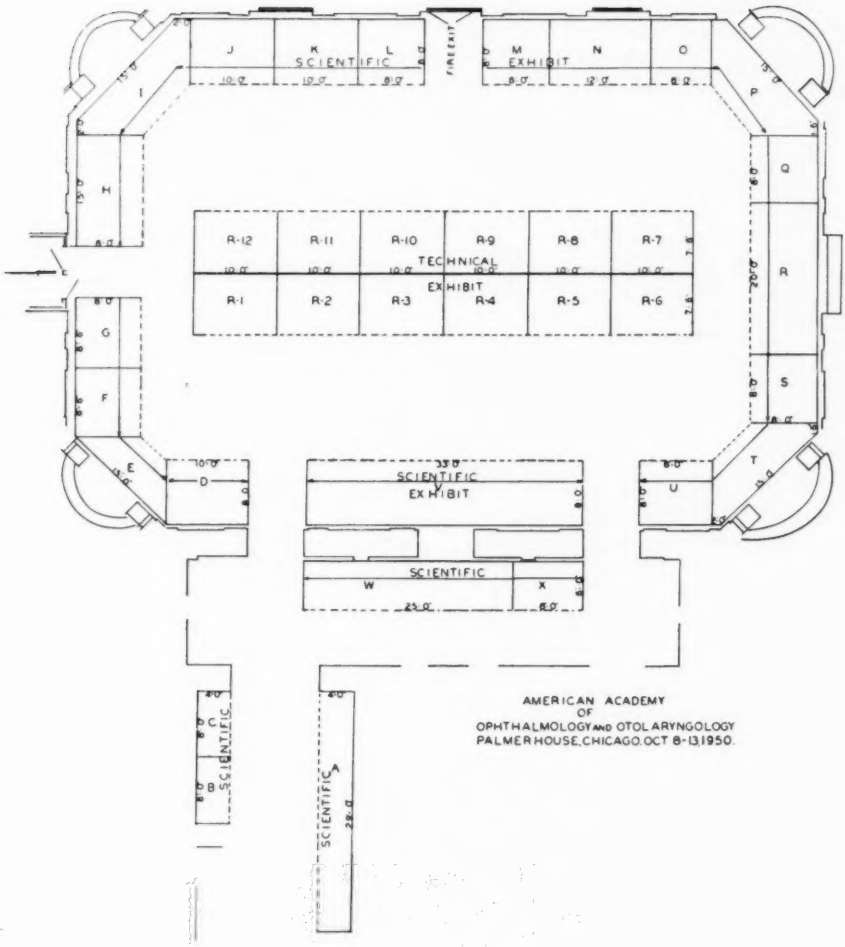
Management of Food Allergy
Herbert J. Rinkel, M.D., Kansas City, Mo.

SCIENTIFIC EXHIBITS

RED LACQUER ROOM

Kenneth L. Roper, *Chairman*

Chicago, Ill.



SPACE A

HOME STUDY COURSES AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Exhibitor:
Lawrence R. Boies, M.D.
Secretary for Home Study Courses

An exhibit of the lessons in ophthalmology and otolaryngology.

SPACE B

AMERICAN BOARD OF OPTICIANRY

Exhibitor:
Albert L. Anderson
BY INVITATION
American Board of Opticianry
Chicago, Ill.

Consists of charts and diagrams concerning the purposes, functions and work of the American Board of Opticianry.. The charts and diagrams contain information concerning (1) educational requirements, curricula and schools of opticianry accredited by the Board, and (2) certification by the Board through examinations for Associate in and Master of Ophthalmic Optics.

SPACE C

PROGRESS AND PROBLEMS IN PREVENTION OF BLINDNESS

Exhibitor:
Franklin M. Foote, M.D.
BY INVITATION
National Society for the
Prevention of Blindness
New York, N. Y.
Co-Exhibitor:
Virginia Smith Boyce
BY INVITATION

Progress has been made in preventing blindness among children due to ophthalmia neonatorum, venereal disease and accidental injury. Many technics in eye surgery have been perfected; for example, cataract operations are 90 to 95 per cent successful. Counterbalancing these gains are new threats to vision. Twenty years have been added to the average life span since 1900. Thus more people develop glaucoma and other blinding diseases that are more prevalent in later years. Recent increases in blindness from hereditary and congenital causes are due partly to the fact that more premature babies—whose eyes are imperfectly developed—are being saved. The National Society is working for the widest and most effective use of the sight-saving knowledge we now have. At the same time it is promoting further research into the causes of blindness and studies on better methods of diagnosis and treatment of eye diseases.

SPACE D

NASOLACRIMAL DUCT: DEVELOPMENTAL ANATOMY

Exhibitor:
J. V. Cassady, M.D.
South Bend, Ind.
Co-Exhibitor:
John M. Thompson, M.D.
BY INVITATION

The developmental anatomy of the nasolacrimal duct is presented in photomicrographs of human embryo sections. At its inception a thickening of the surface ectoderm of the naso-optic fissure detaches itself to become an epithelial cord, buried in the mesenchyme. This cord extends upward and downward, the upper end dividing to form the upper and lower canaliculi, the lower end contacting the nasal mucosa of the inferior meatus. Canalization occurs, the upper end becoming patent early while the lower end, although canalized, is separated from the inferior meatus by a persistent imperforated membrane until near or after birth. The failure of the duct to be perforated at birth is emphasized.

SPACE E

OFFICE BACTERIOLOGY OF THE EYE

Exhibitor:
Frederick H. Theodore, M.D.
Mount Sinai Hospital
New York, N. Y.

The purpose of this exhibit is to demonstrate the ease and feasibility of performing laboratory procedures in routine office practice and to show their value in the diagnosis and treatment of external diseases of the eye. Special emphasis is placed on the practical aspects of slide diagnosis and culture methods. Numerous color photomicrographs of stained slide preparations obtained from illustrative cases are exhibited in specially designed viewing boxes. Bacteriologic diagnosis is correlated with the use of newer therapeutic agents.

SPACE F
INJURY TO EYES IN MICE FOLLOWING A SINGLE WHOLE BODY EXPOSURE OF 400r X-RAYS AT BIRTH

Exhibitor:
Thelma B. Dunn, M.D.
BY INVITATION
National Cancer Institute
Bethesda, Md.

Co-Exhibitor:
Egon Lorenz, Ph.D.
BY INVITATION

Three-panel folding chart, showing diagrams and photographs to describe lesions developing in the eyes of mice exposed to 400r x-rays at birth.

SPACE G
THE DEVELOPMENT OF THE OPHTHALMOSCOPE

Exhibitor:
Frederick C. Cordes, M.D.
San Francisco, Calif.

Co-Exhibitors:
Michael J. Hogan, M.D.
Orwyn H. Ellis, M.D.

This exhibit includes 18 ophthalmoscopes ranging from an original Helmholtz through the various early developments to the modern ophthalmoscope. It has been arranged in observation of the Helmholtz Centenary.

SPACE H
CANCER OF THE MOUTH

Exhibitor:
Gordon B. New, M.D.
Mayo Clinic
Rochester, Minn.

Co-Exhibitors:
John B. Erich, M.D.
and
K. D. Devine, M.D.
BY INVITATION

This exhibit on malignant lesions of the mouth presents, by means of moulages, colored photographs and photomicrographs, various types of malignant tumors that occur in the oral cavity and the methods of therapy which have been found to give the most effective results at the Mayo Clinic. Specifically, malignant lesions of the lips, cheeks, upper and lower jaws, floor of the mouth and tongue are considered in detail. Special emphasis is placed on carcinomas of the tongue; in this region, malignant tumors are particularly serious because of their tendency to undergo metastasis early. Moreover, malignant lesions at the base of the tongue offer a particularly difficult problem in the point of accessibility to treatment. Resection of the mandible by means of surgical diathermy is demonstrated in moulages, as are methods of reconstructing the lower lip and cheek. The exhibit does not pretend to present inalterable technics, but rather suggests principles and plan of therapy. A number of graphs show a statistical evaluation of the end results of treatment.

SPACE I
ROENTGEN ANATOMY OF THE PETROUS PYRAMID AND MASTOID

Exhibitor:
J. Brown Farrior, M.D.
Tampa, Fla.

This exhibit consists of a series of temporal bones which have been mounted in plastic and sectioned to demonstrate the internal anatomy of the petrous pyramid and mastoid. The specimens are sectioned in various planes to demonstrate the anatomy as it is visualized in clinical roentgenography. The Stenver's or Oblique View, the Law or Lateral View, the Towne or Occipital View and the Basilar or Taylor View are each demonstrated by roentgenograms of serial sections of the temporal bone. These are accompanied by a series of clinical roentgenograms which demonstrate practical application of this anatomy.

SPACE J
TRANSPLANTATION OF THE CORNEA

Exhibitor:
Brendan D. Leahey, M.D.
Massachusetts Eye and Ear Infirmary and
St. John's Hospital
Lowell, Mass.

A series of approximately 60 large-sized kodachrome transparencies illustrating different aspects of corneal transplantation including: (a) types of cornea suitable and unsuitable for transplant; (b) preliminary treatment with emphasis on the technic and results of beta radiation; (c) operative technic (since a case depicted has a mature cataract, pictures are also included of the cataract extrac-

tion six months later); (d) complications; (e) typical "before and after" views; (f) a special section, "Transplants in the Dystrophies."

SPACE K
BILATERAL THYROTOMY FOR CARCINOMA OF THE LARYNX, WITH EXTENDED APPLICATIONS

Exhibitor:
Joseph I. Kemler, M.D.
Baltimore, Md.

Co-Exhibitor:
David Davis, M.D.

Fine colored drawings illustrating larynges suitable for the operation; biopsy with the indirect method; the operation, clarifying the doubtful points and emphasizing the important points to the success of the operation; specimens extirpated by operation; larynges before and after operation. Voice reproducing instrument with the speech of patients after operation.

SPACE L
PHOTOGRAPHY OF THE RED REFLEX

Exhibitor:
Virginia Lubkin, M.D.
New York Eye and Ear Infirmary
New York, N. Y.

Enlargements (4 by 5 inches) of kodachromes taken with the Nordenson camera and demonstrating various types of pathologic change as seen with the ophthalmoscope at the level of the red reflex. Cataractous changes, congenital anomalies such as anaridia and lenticonus, tumor of ciliary body, etc. A useful teaching method for the ophthalmologist and internist. A legally valid method of recording progressive pathology, as in traumatic cataract, and as an objective research recording technic. Careful description is given photographic method used.

SPACE M
STEREOPHOTOGRAPHS OF THE ANTERIOR SEGMENT OF THE EYE

Exhibitor:
David D. Donaldson, M.D.
BY INVITATION
Howe Laboratory of Ophthalmology,
Harvard University Medical School
Boston, Mass.

Co-Exhibitor:
David G. Cogan, M.D.

The exhibit is intended to illustrate the use of stereoscopic photography in recording abnormalities of, and surgical procedures on, the anterior segment of the eye. Both the stereoscopic viewer and stereoscopic projector are utilized in observing the pictures. The exhibit demonstrates the following features in stereoscopic photography: (1) the exact localization of abnormalities in the various transparent media of the eye; (2) the importance of utilization of varying magnifications; (3) the occasional indication for exaggerated stereoscopic (hyperstereoscopic) effect.

SPACE N
PATHOLOGY OF THE EYE IN OLD AGE

Exhibitor:
Abraham L. Kornzweig, M.D.
New York, N. Y.

The material for this exhibit was obtained from postmortem study of 120 eyes. These eyes were all obtained from patients at the Home for Aged and Infirm Hebrews, chiefly between the ages of 70 and 90 years. The usual and common findings in the eyes of old people were classified. The unusual findings were also noted, even if they occurred in only one case. Clinical examinations were done on all of these patients, but this exhibit relates chiefly to the pathologic findings in the eyes at autopsy. All of the usual and many of the unusual findings are shown in this exhibit by a series of colored photomicrographs and an explanatory text.

SPACE O
AN OPHTHALMOTROPE CONSTRUCTED ON A NEW PRINCIPLE

Exhibitor:
J. L. Mims, Jr., M.D.
BY INVITATION
Department of Ophthalmology, Tulane
University School of Medicine
New Orleans, La.

Co-Exhibitor:
William B. Clark, M.D.

The exhibit consists of the ophthalmotrope itself, plus photographs and charts showing its usefulness in demonstrating the simultaneous actions of the extraocular muscles. An indicator panel on the model shows the relative contribution of each muscle to rotation of the eye as the globe of the model is turned in all normal directions of gaze.

SPACE P
GLIOMAS OF THE RETINA

Exhibitor:
William L. Benedict, M.D.
Mayo Clinic
Rochester, Minn.

Co-Exhibitors:
Edith M. Parkhill, M.D.
BY INVITATION
Joseph J. Groom, M.D.
BY INVITATION

The exhibit is based on the review of 95 cases of glioma of the retina seen at the Mayo Clinic. It deals with incidence, gross and microscopic features, prognosis and hereditary factors involved in the occurrence of retinal glioma.

SPACE Q
THE TREATMENT OF TUBERCULOSIS OF THE LARYNX BY CHEMOTHERAPY

Exhibitor:
Linden J. Wallner, M.D.
Municipal Tuberculosis Sanitarium of Chicago, Hines Hospital, and University of Illinois
Chicago, Ill.

Co-Exhibitors:
George C. Turner, M.D.
BY INVITATION
Meyer R. Lichtenstein, M.D.
BY INVITATION
Henry C. Sweany, M.D.
BY INVITATION

The exhibit consists of (1) a chart showing the results of treatment by chemotherapy in 122 patients with tuberculosis of the larynx, and the conclusions drawn from this experience; (2) more detailed results in 12 patients. This consists of 4 by 5 inch reproductions of the roentgenograms of the chest before and after treatment, as well as 4 by 5 inch kodachrome enlargements of the mirror appearance of the larynx, before and after therapy. A composite graph of the other indicators, such as temperature, sputum and weight, is also used.

SPACE R
1. LESIONS AND TUMORS OF THE LARYNX

Exhibitor:
Lt. Col. Joseph L. Bernier, D.C.
BY INVITATION
Armed Forces Institute of Pathology
Washington, D. C.

The material in this exhibit includes color transparencies of the benign and malignant tumors of the larynx.

2. NEMATODE ENDOPHTHALMITIS

Exhibitor:
Helenor Campbell Wilder
Armed Forces Institute of Pathology
Washington, D. C.

Co-Exhibitor:
Lawrence P. Ambrogi
BY INVITATION

The role of nematodes in endogenous endophthalmitis, particularly in "pseudoglioma," is demonstrated by color transparencies, plastic models and charts. Nematode larvae within the globe and the reactions which they evoke in this location are illustrated by photomicrographs.

SPACE S
CLINICAL PHOTOGRAPHY IN EAR, NOSE AND THROAT

Exhibitors:
Francis L. Lederer, M.D.
and Associates
Department of Otolaryngology
University of Illinois and Illinois Eye and Ear Infirmary
Chicago, Ill.

Color transparencies covering the field of ear, nose and throat are shown. These include photographs of the ear drum, larynx, intraoral pathology and various other clinical conditions. There are also presented photomicrographs of pertinent pathology. The equipment with which these were taken is demonstrated and the technic explained.

SPACE T
ASSESSMENT OF HEARING BY SPEECH TESTS

Exhibitor:
Theodore E. Walsh, M.D.
Department of Otolaryngology, Washington University School of Medicine and Central Institute for the Deaf
St. Louis, Mo.

Co-Exhibitors:
Hallowell Davis, M.D.
BY INVITATION
S. R. Silverman, Ph.D.
BY INVITATION

The exhibits illustrate the purposes of speech tests and the principles underlying their construction. Application of these principles is illustrated by specific

cases from our clinical series. Apparatus is on display for actual demonstration on visitors. Explanation of the Social Adequacy Index for Hearing is included.

SPACE U
EPIVASCULAR CHOROIDAL PIGMENT STREAKS (SIEGRIST STREAKS)

Exhibitor:
Roy O. Scholz, M.D.
Wilmer Ophthalmological Institute,
Johns Hopkins Hospital
Baltimore, Md.

The epivascular choroidal pigment streaks which Siegrist first described are seldom mentioned in the British and American literature. The cases reported in the literature and the author's own cases have been collected and analyzed. The exhibit is concerned with the clinical description, microscopic and physiologic pathology and prognostic significance of the lesion.

SPACE V
SPEECH AND HEARING REHABILITATION

Exhibitor:
Lawrence J. Linck
BY INVITATION
National Society for Crippled Children and Adults, Inc.
Chicago, Ill.

Co-Exhibitors:
American Speech and Hearing Association
BY INVITATION
University of Iowa
BY INVITATION
Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology

This exhibit consists of four booths, each dealing with an aspect of speech and hearing therapy: (1) cerebral palsy; (2) cleft palate; (3) the preschool deaf or hard-of-hearing child; (4) the school-age child, and the adult having hearing loss. In each booth a physician specializing in the management of the particular speech and hearing defect and a speech and hearing clinician or audiologist will demonstrate the appropriate methods of examination and therapy with patients suffering from the condition under consideration.

SPACE W
STATISTICAL ILLUSTRATIONS FOR OPHTHALMOLOGIC PRESENTATIONS

Exhibitor:
Col. Victor A. Byrnes (MC)
School of Aviation Medicine
Randolph Field, Texas

A presentation using kodachrome transparencies, charts and models showing how certain types of statistical data may be presented in an ophthalmologic lecture or written article to enable the reader or listener to comprehend it readily. Contrasts are made between tabular and visual methods of presenting material. Plans have been made to use a projector to show the difference between the usual typewriter prepared slide and one properly prepared by drawing and photographic reproduction.

SPACE X
ORTHOPTIC EXHIBIT

Exhibitor:
Dolores Engel
BY INVITATION
American Association of Orthoptic Technicians

Co-Exhibitors:
Anita Bargmann, Ruth Fisher, Elizabeth Goggin, Mary Hempstead, Georgia Clausen, and Priscilla Allen
BY INVITATION

The orthoptic exhibit attempts to give an over-all picture of orthoptic clinics throughout the states by means of a large mural. On exhibit also are slides and gadgets made by the technicians for clinic and home use. Orthoptic reprints and a brochure for parents explaining strabismus and its treatment are available at the booth. A technician is present to answer questions or explain orthoptic procedures.

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1896—	Adolph Alt St. Louis, Mo.	W. C. Pipino Des Moines, Iowa	W. L. Dayton Lincoln, Neb.	Hal Foster Kansas City, Mo.	Kansas City
1897—	Adolph Alt St. Louis, Mo.	W. C. Pipino Des Moines, Iowa	W. L. Dayton Lincoln, Neb.	Hal Foster Kansas City, Mo.	St. Louis
1898—	B. E. Fryer Kansas City, Mo.	J. E. Colburn Chicago, Ill.	W. L. Dayton Lincoln, Neb.	Hal Foster Kansas City, Mo.	Chicago
1899—	J. E. Colburn Chicago, Ill.	W. Scheppegrell St. Louis, Mo.	W. L. Dayton Lincoln, Neb.	Thomas A. Woodruff Chicago, Ill.	New Orleans
1900—	W. Scheppegrell St. Louis, Mo.	M. A. Goldstein St. Louis, Mo.	W. L. Dayton Lincoln, Neb.	Fayette C. Ewing St. Louis, Mo.	St. Louis
1901—	M. A. Goldstein St. Louis, Mo.	H. W. Wurdemann Milwaukee, Wis.	W. L. Dayton Lincoln, Neb.	W. L. Ballenger Chicago, Ill.	Cincinnati
1902—	C. R. Holmes Cincinnati, Ohio	W. L. Dayton Lincoln, Neb.	Otto Stein Chicago, Ill.	W. L. Ballenger Chicago, Ill.	Chicago
1903—	W. L. Ballenger Chicago, Ill.	C. J. Stillson Indianapolis, Ind.	Otto Stein Chicago, Ill.	Derrick Vail Cincinnati, Ohio	Indianapolis
1904—	Edward Jackson Denver, Colo.	Dudley Reynolds Louisville, Ky.	Otto Stein Chicago, Ill.	Derrick Vail Cincinnati, Ohio	Denver
1905—	Hanau Loeb St. Louis, Mo.	Derrick Vail Cincinnati, Ohio	Otto Stein Chicago, Ill.	George F. Suker Chicago, Ill.	Buffalo
1906—	Casey Wood Chicago, Ill.	J. A. Stucky Lexington, Ky.	Otto Stein Chicago, Ill.	George F. Suker Chicago, Ill.	St. Clair

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1907—	J. A. Stucky Lexington, Ky.	R. A. Reeve Toronto, Canada	Otto Stein Chicago, Ill.	George F. Suker Chicago, Ill.	Louisville
1908—	Derrick Vail Cincinnati, Ohio	Joseph Beck Chicago, Ill.	Otto Stein Chicago, Ill.	George F. Suker Chicago, Ill.	Cleveland
1909—	Otto Stein Chicago, Ill.	Percy Fridenberg New York, N. Y.	S. H. Large Cleveland, Ohio	George F. Suker Chicago, Ill.	New York
1910—	Wendell Reber Philadelphia, Pa.	L. B. Lockhard Denver, Colo.	S. H. Large Cleveland, Ohio	George F. Suker Chicago, Ill.	Cincinnati
1911—	J. J. Kyle Indianapolis, Ind.	F. Parke Lewis Buffalo, N. Y.	S. H. Large Cleveland, Ohio	George F. Suker Chicago, Ill.	Indianapolis
1912—	George F. Suker Chicago, Ill.	J. W. Murphy Cincinnati, Ohio	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Niagara Falls, Ont.
1913—	J. W. Murphy Cincinnati, Ohio	J. O. McReynolds Dallas, Texas	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Chattanooga
1914—	J. M. Ray Louisville, Ky.	J. M. Ingersoll Cleveland, Ohio	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Boston
1915—	Joseph Beck Chicago, Ill.	Robert S. Lamb Washington, D.C.	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Chicago
1916—	J. E. Brown Columbus, Ohio	T. E. Carmody Denver, Colo.	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Memphis
1917—	W. L. Dayton Lincoln, Neb.	Edward Stieren Pittsburgh, Pa.	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Pittsburgh
1918—	Allen Greenwood Boston, Mass.	Virginius Dabney Washington, D.C.	S. H. Large Cleveland, Ohio	L. M. Francis Buffalo, N. Y.	Denver

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1919	J. M. Ingersoll Cleveland, Ohio	Melville M. Black Denver, Colo.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Cleveland
1920	L. M. Francis Buffalo, N. Y.	Hal Foster Kansas City, Mo.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Kansas City
1921	Emil Mayer New York, N. Y.	John R. Newcomb Indianapolis, Ind.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Philadelphia
1922	Walter R. Parker Detroit, Mich.	Ross H. Skillern Philadelphia, Pa.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Minneapolis and St. Paul
1923	T. E. Carmody Denver, Colo.	Hunter H. McGuire Winchester, Va.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Washington, D. C.
1924	W. B. Lancaster Boston, Mass.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Montreal
1925	Horace Newhart Minneapolis, Minn.	John M. Wheeler New York, N. Y.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Chicago
1926	E. C. Ellett Memphis, Tenn.	Austin A. Hayden Chicago, Ill.	S. H. Large Cleveland, Ohio	Luther C. Peter Philadelphia, Pa.	Colorado Springs
1927	Ross Hall Skillern Philadelphia, Pa.	H. W. Woodruff Joliet, Ill.	S. H. Large Cleveland, Ohio	William P. Wherry Omaha, Neb.	Detroit
1928	Luther C. Peter Philadelphia, Pa.	Frank L. Dennis Colorado Springs, Colo.	S. H. Large Cleveland, Ohio	William P. Wherry Omaha, Neb.	St. Louis
1929	Harris P. Mosher Boston, Mass.	Frank E. Burch St. Paul, Minn.	S. H. Large Cleveland, Ohio	William P. Wherry Omaha, Neb.	Atlantic City
1930	William H. Wilder Chicago, Ill.	Frank R. Spencer Boulder, Colo.	S. H. Large Cleveland, Ohio	William P. Wherry Omaha, Neb.	Chicago

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Ex. Sec'y-Treas.	Comptroller	Meeting Place
1931	John F. Barnhill Miami Beach, Fla.	Meyer Wiener Coronado, Calif.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	French Lick, Ind.
1932	S. Hanford McKee Montreal, Canada	Gordon B. New Rochester, Minn.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Montreal
1933	Burt R. Shurly Detroit, Mich.	Nelson Miles Black Miami, Fla.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Boston
1934	John M. Wheeler New York, N. Y.	LeRoy A. Schall Boston, Mass.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Chicago
1935	Wells P. Eagleton Newark, N. J.	W. Thornwall Davis Washington, D.C.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Cincinnati
1936	Frank E. Burch St. Paul, Minn.	Gordon F. Harkness Davenport, Iowa	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	New York
1937	Lee W. Dean St. Louis, Mo.	Bernard Samuels New York, N. Y.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Chicago
1938	Harry S. Gradle Chicago, Ill.	E. M. Seydell Wichita, Kan.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Washington, D. C.
1939	George M. Coates Philadelphia, Pa.	William W. Pearson Des Moines, Iowa	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Chicago
1940	Frank E. Brawley Chicago, Ill.	Arthur W. Proetz St. Louis, Mo.	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Cleveland
1941	Frank R. Spencer Boulder, Colo.	Everett L. Goar Houston, Texas	William P. Wherry Omaha, Neb.	S. H. Large Cleveland, Ohio	Chicago
1942	Ralph I. Lloyd Brooklyn, N. Y.	Walter H. Theobald Chicago, Ill.	William L. Benedict Rochester, Minn.	S. H. Large Cleveland, Ohio	Chicago
1943	James A. Babbitt Philadelphia, Pa.	Sanford R. Gifford Chicago, Ill.	William L. Benedict Rochester, Minn.	S. H. Large Cleveland, Ohio	Chicago

Date	President	First Vice-President	Ex. Sec'y-Treas.	Meeting Place
1944	Lawrence T. Post St. Louis, Mo.	C. Stewart Nash Rochester, N. Y.	William L. Benedict Rochester, Minn.	Chicago
1945	Gordon B. New Rochester, Minn.	Edmund B. Spaeth Philadelphia, Pa.	William L. Benedict Rochester, Minn.	Meeting Cancelled
1946	Gordon B. New Rochester, Minn.	Edmund B. Spaeth Philadelphia, Pa.	William L. Benedict Rochester, Minn.	Chicago
1947	Alan C. Woods Baltimore, Md.	Arthur C. Jones Boise, Idaho	William L. Benedict Rochester, Minn.	Chicago
1948	Carl H. McCaskey Indianapolis, Ind.	Ralph O. Rychener Memphis, Tenn.	William L. Benedict Rochester, Minn.	Chicago
1949	Conrad Berens New York, N. Y.	Fred W. Dixon Cleveland, Ohio	William L. Benedict Rochester, Minn.	Chicago

SECTION ON INSTRUCTION

FIFTY-FIFTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 8-13, 1950
PALMER HOUSE
Chicago, Illinois

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1950 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $1.50. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

Refunds will be granted only if request for reimbursement is received prior to October 6.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to September 25, send orders to 100 First Avenue Building, Rochester, Minnesota; after September 25, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois.

Ordering Instruction Tickets

An *individual* course is complete in 1 period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts 2 or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited it is necessary that you*

select more courses than it is possible for you to attend; in other words, a list of three preferences for each period selected should be given.

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. In each case, every effort is made to include first choices. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Ophthalmology, forenoons, periods 1-2-3 — Otolaryngology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday	(F)riday
Period a.m.		1 2 3	1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6	

M-4	2:00 p.m.	to 3:00 p.m.	W-5	3:15 p.m.	to 4:15 p.m.
M-5	3:15 p.m.	to 4:15 p.m.	W-6	4:30 p.m.	to 5:30 p.m.
M-6	4:30 p.m.	to 5:30 p.m.	Th-1	9:00 a.m.	to 10:00 a.m.
T-1	9:00 a.m.	to 10:00 a.m.	Th-2	10:15 a.m.	to 11:15 a.m.
T-2	10:15 a.m.	to 11:15 a.m.	Th-3	11:30 a.m.	to 12:30 p.m.
T-3	11:30 a.m.	to 12:30 p.m.	Th-4	2:00 p.m.	to 3:00 p.m.
T-4	2:00 p.m.	to 3:00 p.m.	Th-5	3:15 p.m.	to 4:15 p.m.
T-5	3:15 p.m.	to 4:15 p.m.	Th-6	4:30 p.m.	to 5:30 p.m.
T-6	4:30 p.m.	to 5:30 p.m.	F-1	9:00 a.m.	to 10:00 a.m.
W-1	9:00 a.m.	to 10:00 a.m.	F-2	10:15 a.m.	to 11:15 a.m.
W-2	10:15 a.m.	to 11:15 a.m.	F-3	11:30 a.m.	to 12:30 p.m.
W-3	11:30 a.m.	to 12:30 p.m.			
W-4	2:00 p.m.	to 3:00 p.m.			

(Familiarity with this key will be helpful and will avoid confusion)

CORRECTIONS

Ophthalmology

The title of Continuous Course No. 8, presented by Dr. Frederick C. Cordes, has been changed to **Anomalies of the Optic Disc.**

Course No. 216, **End Results in Retinal Detachment Surgery,** to have been presented by Dr. Edward P. Burch, has been cancelled.

Otolaryngology

The continuous course in **Practical Audiometry** given by Dr. Werner Mueller was erroneously listed on the chart in the March-April issue of the TRANSACTIONS. This course will not be given on Monday. As Course 144 this lecture will be presented in Room 731 on Tuesday, periods 4 and 5; as Course 145 it will be presented in Room 795 on Wednesday, periods 4 and 5.

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $1.50 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to SEPTEMBER 25

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
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ADVANCE ORDER SHEET

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NO REFUNDS WILL BE MADE AFTER OCTOBER 6

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday
- ☐ Friday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, Thursday and Friday are represented by the letters M, T, W, Th, F. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All ophthalmologic courses are presented in the mornings. All courses in otolaryngology and maxillofacial surgery are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1950 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
FRI. 1						
A.M. 2						
3						

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED
1 or 2	Allen, J.	Bacteriology	2 PERIODS T-1, T-2 or W-1, W-2
3	Bedell	.....SOLD OUT.....	
4	Berliner	.....SOLD OUT.....	
5	Brown, et al	Eye Muscles	8 PERIODS T-1, T-2, W-1, W-2, Th-1, Th-2, F-1, F-2
6 or 7	Copeland	Streak Retinoscopy	4 PERIODS T-1, T-2, W-1, W-2 or Th-1, Th-2, F-1, F-2
8	Cordes	Anomalies of the Optic Disc	2 PERIODS T-1, T-2
9	Cordes	Practical Observations in Refraction	2 PERIODS Th-1, Th-2
10	Costenbader and Swan	Binocular Vision	9 PERIODS T-2, T-3, W-2, W-3, Th-2, Th-3, F-1, F-2, F-3
11	Dunnington and Wheeler	.....SOLD OUT.....	
12	Fralick	Applied Anatomy	4 PERIODS T-1, W-1, Th-1, F-1
13	Gordon	Perimetry	4 PERIODS T-3, W-3, Th-3, F-3
14 or 15	Haas and McGarry	Introduction to Gonioscopy	2 PERIODS Th-2, Th-3 or F-2, F-3
16	Hastings	Psychosomatic Considerations	2 PERIODS Th-1, Th-2
17	Henderson	Neurologic Applied Anatomy	3 PERIODS T-3, W-3, Th-3
18	Hughes, et al	Plastic Surgery	8 PERIODS T-2, T-3, W-2, W-3, Th-2, Th-3, F-2, F-3
19	Jardon	Fitting of Ocular Prostheses	2 PERIODS W-2, W-3
20	Knighton, et al	Glaucoma	4 PERIODS T-1, W-1, Th-1, F-1
21	Kuhn	Industrial Ophthalmology	3 PERIODS T-1, W-1, Th-1
22	McLean, et al	.....SOLD OUT.....	
23	Meyer, et al	Glaucoma	8 PERIODS T-2, T-3, W-2, W-3, Th-2, Th-3, F-2, F-3
24	Newell	Rabbit Surgery	2 PERIODS F-1, F-2
25	O'Rourke	Manifest and Cycloplegic Refraction	2 PERIODS W-1, W-2
26	Payne, et al	Special Eye Pathology	4 PERIODS T-1, T-2, T-3, T-3a
27	Regan	Refraction	12 PERIODS T-1, T-2, T-3, W-1, W-2, W-3, Th-1, Th-2, Th-3, F-1, F-2, F-3
28	Rones and Stow	Clinical Pathology	8 PERIODS T-2, T-3, W-2, W-3, Th-2, Th-3, F-2, F-3
29	Scarney and Johnson	Slit Lamp Biomicroscopy	8 PERIODS T-2, T-3, W-2, W-3, Th-2, Th-3, F-2, F-3
30	Scobee, et al	.....SOLD OUT.....	
31	Sheard	Applied Physiologic Optics	4 PERIODS T-3, W-3, Th-3, F-3
32	Sheppard	Vertical Deviations	2 PERIODS T-1, T-2
33	Theobald, et al	Histopathology	12 PERIODS T-1, T-2, T-3, W-1, W-2, W-3, Th-1, Th-2, Th-3, F-1, F-2, F-3
34	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS T-1, T-2, T-3, W-1, W-2, W-3, Th-1, Th-2, Th-3, F-1, F-2, F-3
35	Wiener	.....SOLD OUT.....	

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Trends in Cataract Surgery
207	Baker	Neuralgias of the Head
209	Berens	Ophthalmic Surgery
213	Boeder	Studies in Visual Space
217	Byrnes	Visual Problems of Supersonic Speeds
231	Davies	Pathology of Iris & Ciliary Body
235	Dunlap	Anomalies of Divergence
237	Elwyn	Fundus Changes in Hypertension
239	Falls	Ocular Geriatrics
247	Gipner	Recent Advances
258	Irvine	Uveitis
270	Lloyd	Corneal Lesions
274	Mann, M.	Hypertensive Changes in Fundus
284	Ogle	Problems of Binocular Vision
285	Owens & Owens	Retrolental Fibroplasia
289	Pfeiffer	Diagnosis with Aid of X-ray
297	Scheie	Traumatic Surgery
306	Tait	Accommodation & Convergence Reflexes
309	Troncoso	Clinical Application of Gonioscopy
A	Home Study	Anatomy

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
202	Alvaro	Radium & X-ray
218	Calhoun	Corneal Lesions
223	Clark	Iridectomy
228	Culler	Chiasmal Syndromes
232	Dickey	Hypertropia
244	Gailey	Surgery for Cataract Patient
250	Gray	Intraocular Foreign Bodies
257	Iliff	.....SOLD OUT.....
268	Lepard	Eye Diseases Common to Children
275	Mann, W.	Refraction for Cataract Patient
286	Paton	Corneal Surgery
290	Pino	Problems of Bifocal Lenses
296	Rychener	External Diseases
298	Simpson	Office Management of Phorias
302	Stine	Retinal Detachment
314	von Sallmann	Antibiotics
318	Woods	Ocular Tuberculosis
B	Home Study	Embryology

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
206	Baird	Acute Lesions of Cornea
211	Bettman	Orbital Lesions
215	Braley	Virus Diseases
219	Callahan	Repair of Lid Lacerations
222	Chandler	Glaucoma in Aphakia
227	Cruthirds	Burns of the Eye
238	Elwyn	Heredodegenerative Diseases of Retina
245	Gemeroy	External Diseases
254	Gump	Iridocyclitis
255	Harrington	Ocular Neuroses
259	Jobe	Practical Physiologic Optics
276	Marshall	Chronic Infections of Lid
278	McGuire	Surgery of Vertical Muscles
287	Perera	End Results in Retinal Detachment
293	Robinson	Retarded Reader & Causes
295	Rundles	Streak Retinoscopy
301	Sterling	Practical Optics
307	Thygeson	Herpetic Viruses
308	Town	Fibrin Coagulum in Ocular Surgery
C	Home Study	Physiology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Trends in Cataract Surgery
207	Baker	Neuralgias of the Head
224	Cooper	..... SOLD OUT .....
229	Cusick	Ptosis
231	Davies	Pathology of Iris & Ciliary Body
235	Dunlap	Anomalies of Divergence
240	Falls	Practical Application of Heredity
241	Finegan	Ocular Tuberculosis
247	Gipner	Recent Advances
258	Irvine	Uveitis
271	Lloyd	Retina & Cerebral Representation
272	Macnie	Eye Changes in Head Trauma
274	Mann, M.	Hypertensive Changes in Fundus
284	Ogle	Problems of Binocular Vision
285	Owens & Owens	Retrolental Fibroplasia
289	Pfeiffer	Diagnosis with Aid of X-ray
292	Reese	Tumors of Eye & Adnexa
297	Scheie	Traumatic Surgery
305	Swan	Therapeutic Principles
306	Tait	Accommodation & Convergence Reflexes
309	Troncoso	Clinical Application of Gonioscopy
D	Home Study	Optics

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
202	Alvaro	Radium & X-ray
218	Calhoun	Corneal Lesions
223	Clark	Iridectomy
228	Culler	Chiasmal Syndromes
232	Dickey	Hypertropia
243	Friedenwald	Problems in Tonometry
244	Gailey	Surgery for Cataract Patient
251	Guibor	Physiology of Ocular Muscles
257	Iliff	Beta Irradiation Therapy
268	Lepard	Eye Diseases Common to Children
275	Mann, W.	Refraction for Cataract Patient
279	McLaughlin	Chemical Eye Injuries
286	Paton	Corneal Surgery
291	Pino	Increasing Eye Care
296	Rychener	External Diseases
298	Simpson	Office Management of Phorias
302	Stine	Retinal Detachment
314	von Sallmann	Antibiotics
E	Home Study	Refraction

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
206	Baird	Acute Lesions of Cornea
211	Bettman	Orbital Lesions
215	Braley	Virus Diseases
219	Callahan	Repair of Lid Lacerations
227	Cruthirds	Burns of the Eye
245	Gemeroy	External Diseases
254	Gump	Iridocyclitis
255	Harrington	Ocular Neuroses
259	Jobe	Practical Physiologic Optics
262	Kronfeld	Retinal Detachment
276	Marshall	Chronic Infections of Lid
280	Meek	Surg. Anatomy of Orbit
287	Perera	End Results in Retinal Detachment
293	Robinson	Retarded Reader & Causes
295	Rundles	Streak Retinoscopy
300	Spaeth	Lacrimal Gland & Sac
301	Sterling	Practical Optics
307	Thygeson	Herpetic Viruses
308	Town	Fibrin Coagulum in Ocular Surgery
F	Home Study	External Diseases & Bacteriology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
203	Anthony	The Fractured Orbit
210	Berke	.....SOLD OUT.....
214	Boeder	Aniseikonia
216	Burch	..... CANCELLED .....
224	Cooper	Convergent Strabismus in Children
225	Cosgrove	Subacute Conjunctivitis
226	Cowen	Acute Orbital Infections
229	Cusick	Ptosis
234	Duane	Anomalous Retinal Correspondence
236	Dunnington	End Results in Muscle Surgery
248	Givner	Systemic Diseases
261	Klien	Pathology of Retinal Lesions
263	Lancaster	.....SOLD OUT.....
267	Lemoine	Vitreous Opacities
277	McCaslin	Anesthesia
282	Mullen	Ophthalmic Emergencies
288	Perritt	Advances in Corneal Surgery
303	Sugar	Primary Atrophy
305	Swan	Therapeutic Principles
313	Veasey	Refraction Difficulties
315	Weisser	Chronic Uveitis
G	Home Study	Perimetry

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
201	Allen, T.	Office Practice
204	Ascher	Aqueous Veins
220	Carter	Vascular Diseases of Fundus
221	Cassady	Dacryocystitis of Infancy
233	Droege-mueller	Refinements in Refraction
246	Gifford	Dacryocystorhinostomy
252	Guibor	Neurology of Ocular Muscles
256	Hester	Clinical Photography
266	Leinfelder	Papilledema & Optic Neuritis
269	Linksz	Ocular Therapeutics
273	Mangold	Proper Fitting of Glasses
279	McLaughlin	Chemical Eye Injuries
294	Roper	Ocular Chemotherapy
299	Snydacker	Hyperphoria
304	Sullivan	Practical Perimetry
310	Trueman	Demyelinizing Diseases
316	Whalman	Surgical Procedures
H	Home Study	Extraocular Muscles

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Senile Changes in Crystalline Lens
212	Birge	Ocular Mycology
242	Fink	Surg. Anatomy of Oblique Muscles
249	Glover	Office Treatment
260	Katz	Corneal Dystrophies
264	Leahey	External Ocular Malignancy
265	Lebensohn	Presbyopia
278	McGuire	Surgery of Vertical Muscles
280	Meek	Surg. Anatomy of Orbit
281	Morrison	Office Management of Glaucoma
283	Nugent	Contact Lenses
300	Spaeth	Lacrimal Gland & Sac
311	Vail	Operation of Choice in Cataract Surgery
317	Wood	Macular Lesions
319	Yudkin	Deficiency Diseases
I	Home Study	Ophthalmoscopy & Slit Lamp

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Friday

Key F-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
203	Anthony	The Fractured Orbit
210	Berke	.....SOLD OUT.....
216	Burch	..... CANCELLED .....
225	Cosgrove	Subacute Conjunctivitis
226	Cowen	Acute Orbital Infections
230	Cutler	Enucleation
234	Duane	Anomalous Retinal Correspondence
241	Finegan	Ocular Tuberculosis
248	Givner	Systemic Diseases
261	Klien	Pathology of Retinal Lesions
263	Lancaster	The Red-Green Test
267	Lemoine	Vitreous Opacities
277	McCaslin	Anesthesia
282	Mullen	Ophthalmic Emergencies
288	Perritt	Advances in Corneal Surgery
312	Vail	Cataract Extraction by Smith Method
313	Veasey	Refraction Difficulties
315	Weisser	Chronic Uveitis
J	Home Study	Pathology

Key F-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
204	Ascher	Aqueous Veins
220	Carter	Vascular Diseases of Fundus
221	Cassady	Dacryocystitis of Infancy
233	Droege-mueller	Refinements in Refractions
246	Gifford	Dacryocystorhinostomy
253	Guibor	Neuromotor Defects of Ocular Muscles
256	Hester	Clinical Photography
266	Leinfelder	Papilledema & Optic Neuritis
269	Linksz	Ocular Therapeutics
273	Mangold	Proper Fitting of Glasses
294	Roper	Ocular Chemotherapy
299	Snydacker	Hyperphoria
304	Sullivan	Practical Perimetry
310	Trueman	Demyelinizing Diseases
316	Whalman	Surgical Procedures
K	Home Study	Pharmacology & Therapeutics

Key F-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Senile Changes in Crystalline Lens
212	Birge	Ocular Mycology
242	Fink	Surg. Anatomy of Oblique Muscles
249	Glover	.....SOLD OUT.....
264	Leahey	External Ocular Malignancy
265	Lebensohn	Presbyopia
281	Morrison	.....SOLD OUT.....
283	Nugent	Contact Lenses
317	Wood	Macular Lesions
319	Yudkin	Deficiency Diseases

CONTINUOUS COURSES — OTOLARYNGOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED
101	Altmann and Waltner	Histopathology of Ear	4 PERIODS W-4, W-5, Th-4, Th-5
102 or 103	Baker, D. C.	Applied Anatomy of Larynx	2 PERIODS M-5, M-6 or T-5, T-6
104	Baker, H. K.	Organic Speech Disorders	2 PERIODS T-5, T-6
105 or 106	Blocker	Principles of Plastic Surgery	2 PERIODS W-4, W-5 or Th-4, Th-5
107 or 108	Bordley	Deafness in Preschool Child	2 PERIODS M-5, M-6 or T-5, T-6
109 or 110	Campbell	Hearing Disorders	2 PERIODS W-4, W-5 or Th-4, Th-5
111 or 112 or 113	Clerf	Paralysis of Larynx	2 PERIODS M-4, M-5 or T-4, T-5 or W-5, W-6
114 or 115	Conley	Cancer of Head & Neck	2 PERIODS W-5, W-6 or Th-5, Th-6
116	Converse	.....SOLD OUT.....	
117 or 118	Cullen	Safe Anesthesia	2 PERIODS M-5, M-6 or T-5, T-6
119 or 120	Figi	Lesions of Mouth	2 PERIODS W-4, W-5 or Th-4, Th-5
121	Fowler, E. and Montreuil	Histology of Ear	4 PERIODS M-4, M-5, T-4, T-5
122 or 123	Goldman	Clinical Bacteriology	2 PERIODS W-4, W-5 or Th-4, Th-5
124 or 125 or 126 or 127	Hansel	Allergy	2 PERIODS M-4, M-5 or T-4, T-5 or W-4, W-5 or Th-4, Th-5
128 or 129	Hastings	Psychosomatic Principles	2 PERIODS T-4, T-5 or Th-4, Th-5
130	Hauser and Jennings	Histopathology	6 PERIODS M-4, M-5, T-4, T-5, W-5, W-6
131	Jackson	Technic in Peroral Endoscopy	3 PERIODS T-4, W-4, Th-4
132 or 133	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS M-5, M-6 or T-5, T-6
134 or 135	Lempert	Modern Temporal Bone Surgery	2 PERIODS M-4, M-5 or W-4, W-5
136 or 137	Lewin	Reconstructive Surgery of External Ear	2 PERIODS W-4, W-5 or Th-4, Th-5
138 or 139	Lindsay	Vertigo	2 PERIODS W-5, W-6 or Th-5, Th-6
140 or 141	Marcus and Utley	The Deaf Child	2 PERIODS M-4, M-5 or T-4, T-5
142 or 143	Martin	Cancer of Larynx	2 PERIODS M-5, M-6 or T-5, T-6
144 or 145	Mueller	Practical Audiometry	2 PERIODS T-4, T-5 or W-4, W-5
146 or 147	Orton	Surgery of Neck	2 PERIODS M-5, M-6 or T-5, T-6
148 or 149	Owen	Attic-Aditus-Antral Area	2 PERIODS M-4, M-5 or T-4, T-5
150 or 151	Scheldrup	Surg. Anatomy of Neck	2 PERIODS M-5, M-6 or T-5, T-6
152	Schlemer	Medicolegal Medicine	2 PERIODS T-4, T-5
153 or 154	Silverman	Clinical Use of Speech Tests	2 PERIODS W-5, W-6 or Th-5, Th-6
155	Slaughter	Plastic Surgery & Growing Child	2 PERIODS M-4, M-5
156	Tucker, et al	Esophageal Disease & Foreign Bodies	3 PERIODS T-4, W-4, Th-4
157	Tucker, et al	Laryngeal Surgery	3 PERIODS M-6, T-6, W-6
158 or 159	Turchik	Rhinoplasty	2 PERIODS M-5, M-6 or T-5, T-6
160 or 161	Wright	Pharmacology of Useful Drugs	2 PERIODS W-5, W-6 or Th-5, Th-6

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Deafness
408	Beck	Maxillary Sinusitis
409	Becker	Plastic Surgery of Protruding Ears
414	Bryant	Hypothyroidism
415	Caron	Collapsing Alae
417	Costen	Mandibular Joint Syndrome
438	Goodhill	.....SOLD OUT.....
439	Goodyear	Hemilaryngectomy
442	Harkness	Signs, Syndromes, & Triads in Private Practice
443	Harris	The Use of Tantalum
446	Hill	Mistakes Made in Otolaryngology
450	Horton	Headaches & Head Pains
453	Hunnicutt	Chronically Congested Nose
455	Jordan	Otitis Media
458	Kemler	Laryngeal Lesions
468	Meltzer	Management of Deafness
469	Miller	Laryngeal Emergencies in Infants & Children
472	Myklebust	Deafness in Young Children
480	Priest	X-ray Diagnosis
492	Silcox	Bacterial Allergy
N	Home Study	Hearing Function

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
403	Ashley	Dust Therapy in Respiratory Allergy
410	Blassingame	Irrigation of Maxillary Sinuses
412	Boyden	Chronic Frontal Sinusitis
418	Craft	Nasal Surgery in Allergic Patient
420	Curtis	Common Oral Lesions
424	Davison	Acute Obstructive Laryngitis in Children
427	DeWeese	.....SOLD OUT.....
433	Forman	Problems in Laryngoscopy
440	Hall	Deep Neck Infections
448	Holinger	Diseases of Esophagus
460	Kuhn	Allergic Diseases of Ear
477	Owen	Respiratory Allergy
494	Spake	Practical Rhinology
503	Mosher	Diseases of the Esophagus
O	Home Study	Vestibular Function

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
413	Boxer	Endoscopic Problems in Children
419	Croushore	Management of Sinusitis
421	Daley	Analysis of Otorhinologic Deformities
422	Davis	.....SOLD OUT.....
429	Eagle	Uncommon Otolaryngologic Syndromes
430	Ersner	Rhinoplastic Problems
432	Fisher	Esophageal Obstruction
434	Fowler, W.M.	Blood Dyscrasias
435	Furstenberg	Organic Neurologic Considerations
444	Hesser	Facial Pains
454	Jones	Ear Problems in Children
462	Lowy	Physiology of Hearing
474	New	Plastic Surgery of Face
481	Proud	Acrylic Implant for Atrophic Rhinitis
482	Reger	Interpretation of Audiograms
489	Seltzer	Plastic Surgery of Nose
499	Wanamaker	Facial Maxillary Surgery
P	Home Study	Physiology

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Deafness
408	Beck	Maxillary Sinusitis
409	Becker	Plastic Surgery of Protruding Ears
414	Bryant	Hypothyroidism
415	Caron	Collapsing Alae
417	Costen	Mandibular Joint Syndrome
438	Goodhill	.....SOLD OUT.....
439	Goodyear	Hemilaryngectomy
442	Harkness	Signs, Snydromes, & Triads in Private Practice
443	Harris	The Use of Tantalum
446	Hill	Mistakes Made in Otolaryngology
450	Horton	Headaches & Head Pains
453	Hunnicutt	Chronically Congested Nose
455	Jordan	Otitis Media
458	Kemler	Laryngeal Lesions
468	Meltzer	Management of Deafness
469	Miller	Laryngeal Emergencies in Infants & Children
472	Myklebust	Deafness in Young Children
481	Proud	Acrylic Implant for Atrophic Rhinitis
492	Silcox	Bacterial Allergy
Q	Home Study	Pharmacology

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
403	Ashley	Dust Therapy in Respiratory Allergy
410	Blassingame	Irrigation of Maxillary Sinuses
412	Boyden	Chronic Frontal Sinusitis
418	Craft	Nasal Surgery in Allergic Patient
420	Curtis	Common Oral Lesions
424	Davison	Acute Obstructive Laryngitis in Children
427	DeWeese	.....SOLD OUT.....
433	Forman	Problems in Laryngoscopy
440	Hall	Deep Neck Infections
448	Holinger	Diseases of Esophagus
460	Kuhn	Allergic Diseases of Ear
477	Owen	Respiratory Allergy
494	Spake	Practical Rhinology
R	Home Study	Bacteriology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
413	Bozer	Endoscopic Problems in Children
419	Croushore	Management of Sinusitis
421	Daley	Analysis of Otorhinologic Deformities
423	Davis	Acute Laryngotracheobronchitis
429	Eagle	Uncommon Otolaryngologic Syndromes
430	Ersner	Rhinoplastic Problems
432	Fisher	Esophageal Obstruction
434	Fowler,W.M.	Blood Dyscrasias
435	Furstenberg	Organic Neurologic Considerations
444	Hesser	Facial Pains
454	Jones	Ear Problems in Children
461	Long	Chemotherapeutic & Antibiotic Agents
462	Lowy	Physiology of Hearing
465	McLaurin	External Otitis
474	New	Plastic Surgery of Face
489	Seltzer	Plastic Surgery of Nose
499	Wanamaker	Facial Maxillary Surgery
S	Home Study	Histology

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
404	Batson	Anatomy of Ear
416	Converse	Surgery of Nasal Deviations
426	Derlacki & Shambaugh	Skin Testing
428	Dingman	Correction of Nasal Deformities
436	Gilbert	Septal Surgery Associated with Deformities
441	Hampsey	History Taking & Prophylaxis in Allergy
445	Hilger	Autonomic Dysfunction
447	Holinger	Bronchoscopic Technics
449	Hoople	Otitis Media with Effusion
451	Horton	Histamine in Ophthalmology & Otolaryngology
459	Kinkade	Acute & Chronic Sinusitis
467	McNaught	Surg. Anatomy & Procedures — Upper Sinuses
473	Nash	Otolaryngologic Mishaps
475	Ogura	One-Stage Widefield Laryngectomy
476	Osterberg	Antihistaminics, etc.
483	Reger	Elementary Otological Acoustics
493	Singleton	Otitis Media with Effusion
495	Thornell	Intralaryngeal Arytenoidectomy
497	Wallner	Indirect Laryngoscopy
501	Williams	Specific Treatment of Allergies
T	Home Study	Anatomy—Ear

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
402	Anthony	Hemorrhage of Nose & Throat
405	Batson	Anatomy of Nose
407	Beavis	Velopharyngeal Reconstruction
411	Boies	Headaches & Neuralgia of Nasal Origin
437	Gill	External Otitis
452	House	Chronically Discharging Ear
457	Keaster	Speech Problems in Children
466	McNally	Labyrinthine Tests
470	Moore	Cysts & Fistulas of Face & Neck
479	Pressman	Problems in Rhinoplasty
480	Priest	X-ray Diagnosis
484	Richards	.....SOLD OUT.....
496	Tremble	Practical Therapeutics
502	Work	Hearing: Basic Principles
U	Home Study	Anatomy—Nasal Space

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
406	Batson	Anatomy of Larynx & Neck
431	Farrior	Primary Skin Graft in Radical Mastoidectomy
456	Juers	Hearing Tests
461	Long	Chemotherapeutic & Antibiotic Agents
463	Marden	Antibiotics & Chemotherapy
464	Maxwell	Facial Paralysis
465	McLaurin	External Otitis
471	Morrison	Tonsillectomy
478	Perlman	Eustachian Tube Disease
485	Roberts	Headache
486	Sahs	Identification of Functional Disorders
487	Salinger	Rhinoplasty
488	Sellers	The Common Cold
490	Senturia	External Otitis
491	Shambaugh	Allergy in Relation to Sinusitis
498	Walsh	Vasomotor Rhinitis & Chronic Sinusitis
500	Warren	Practical Therapeutics
V	Home Study	Anatomy—Pharynx, etc.

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
404	Batson	Anatomy of Ear
426	Derlacki & Shambaugh	Skin Testing
428	Dingman	Correction of Nasal Deformities
436	Gilbert	Septal Surgery Associated with Deformities
441	Hampsey	History Taking & Prophylaxis in Allergy
445	Hilger	Autonomic Dysfunction
447	Holinger	Bronchoscopic Technics
449	Hoople	Otitis Media with Effusion
451	Horton	Histamine in Ophthalmology & Otolaryngology
459	Kinkade	Acute & Chronic Sinusitis
467	McNaught	Surg. Anatomy & Procedures—Upper Sinuses
473	Nash	Otolaryngologic Mishaps
475	Ogura	One-Stage Widefield Laryngectomy
476	Osterberg	Antihistaminics, etc.
483	Reger	Elementary Otological Acoustics
493	Singleton	Otitis Media with Effusion
495	Thornell	Intralaryngeal Arytenoidectomy
497	Wallner	Indirect Laryngoscopy
501	Williams	Specific Treatment of Allergies
W	Home Study	Otological Acoustics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
402	Anthony	Hemorrhage of Nose & Throat
405	Batson	Anatomy of Nose
407	Beavis	Velopharyngeal Reconstruction
411	Boies	Headaches & Neuralgia of Nasal Origin
437	Gill	External Otitis
452	House	Chronically Discharging Ear
457	Keaster	Speech Problems in Children
466	McNally	Labyrinthine Tests
470	Moore	Cysts & Fistulas of Face & Neck
479	Pressman	Problems in Rhinoplasty
484	Richards	Suggestions Concerning Office Practice
496	Tremble	Practical Therapeutics
502	Work	Hearing: Basic Principles
X	Home Study	Pathology

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
406	Batson	Anatomy of Larynx & Neck
431	Farrior	Primary Skin Graft in Radical Mastoidectomy
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491	Shambaugh	Allergy in Relation to Sinusitis
498	Walsh	Vasomotor Rhinitis & Chronic Sinusitis
500	Warren	Practical Therapeutics

TECHNICAL EXHIBITS

The Academy is appreciative of the co-operation of all technical exhibitors.

Booths with numbers preceded by "R" are located in the Red Lacquer Room.

Abbott Laboratories, North Chicago, Ill.
Booths 106 and 107

Alcon Laboratories, Inc., Fort Worth, Texas **Booth 57**

Alcon Laboratories, Incorporated, is featuring nasal and ophthalmic solutions carefully prepared to parallel the tonicity and pH values of the orifices in which they are intended to be used.

Alkalol Co., Taunton, Mass.
Booth 29

The Alkalol Company is featuring ALKALOL, a solution especially designed for the treatment of the mucous membranes of the eye, nose, and throat.

First compounded in 1896, its balanced formula of salts and essential oils makes a solution that is effective and at the same time nontoxic, mild and pleasant. It is alkaline and a mucus solvent.

ALKALOL is an excellent vehicle for many drugs. For further information, please call at Booth 29.

American Cystoscope Makers, Inc., New York, N. Y. **Booths 91 and 92**

American Cystoscope Makers, Inc., extend a cordial invitation to the members and guests of the American Academy of Ophthalmology and Otolaryngology to visit their booth. There will be on display precision telescopic diagnostic instruments including small caliber nasopharyngoscopes, otoscopes, ophthalmoscopes, antroscopes, laryngoscopes, bronchoscopes, esophagoscopes, endoscopic photographic equipment, and allied diagnostic instruments.

American Optical Co., Southbridge, Mass.
Booths 7 thru 13

Members of the American Academy of Ophthalmology and Otolaryngology and their guests are cordially invited to visit the American Optical Company exhibit. There will be on display the latest ophthalmologic instruments and equipment —also some of their scientific instruments. Among the items displayed will be the latest AO Trial Frame, the Space Eikonometer, the Renshaw Stereo Disparator, the improved plastic eye implants and artificial eyes. American Optical Company representatives will be on hand to demonstrate and explain these instruments to those interested.

Associated Medical Care Plans, Chicago, Ill. **Booths R-10 and R-11**

Designed to offer hospitality and courteous service to the doctors, the Blue Shield exhibit portrays a man's den or library. On behalf of the more than 16,000,000 Blue Shield members, it is hoped the doctors will find it a comfortable spot in which to relax for a few minutes and learn the latest developments of these voluntary nonprofit medical care plans.

Barnett Optical Laboratories, Chicago, Ill.
Booth 37

Bausch & Lomb Optical Co., Rochester, N. Y.
Booths 87 thru 90 and 95 thru 98

Featured at the Bausch & Lomb exhibit will be Bausch & Lomb's new Retinal Camera, recently viewed at the International Congress of Ophthalmology in London and seen for the first time at the Academy convention last year. The only modern instrument of its kind available, the camera is now in production and has already been extensively used at Duke University and the U. S. Marine Hospital in Baltimore in connection with the heart-study program sponsored by the U. S. Public Health service.

In addition, the Bauch & Lomb display will comprise the complete line of B&L ophthalmic and diagnostic instruments, including the DeLuxe Refracting Unit, Green's Refractor, Compact Acuity Projector, and various types of slit lamps. Ophthalmic lenses—single vision and multifocal—and latest types of eyewear will also be shown.

Belgard, Inc., Chicago, Ill.
Booths 18 and 19½

Austin Belgard and J. Ralph Lowrey will present the latest developments in refracting and orthoptic adjuncts.

Belgard-Spero, Inc., Chicago, Ill.
Booths 61 and 62

The House of Vision intends to have a most interesting exhibit of unusual, new, English, Swiss and German equipment which should prove of great interest to the medical profession.

We shall display the Aimark Projection Perimeter; the new Aimark Slit-lamp, which embodies a new principle of illumination; the Haag-Streit Goldmann Slit-lamp and Goldmann perimeter; the new Zeiss Comberg Slit-lamp; the Hamblin Lister Perimeter; Hamblin Hand Rotary Prisms; Maddox Wing Test; Aimark Hand Phorometer; Remy Separator; Hand Diploscope; Hamblin Mutch Proptometer; and The House of Vision Distometer.

In addition to these very interesting items of equipment, several other refracting aids will be shown, as well as some unusual lens grinding examples such as are described in the "Scientific Corner" monthly.

Bell & Howell Co., Chicago, Ill.
Booth 58

Bell and Howell will exhibit its complete line of 8 and 16 mm. motion picture cameras and projectors. The exhibit will also feature the new, amazing Bell & Howell 35 mm. still camera, "the Foton," in addition to time and motion study projection equipment. Visit the Bell & Howell booth to see a complete line of motion picture equipment.

Beltone Hearing Aid Co., Chicago, Ill.
Booth R-4

N. P. Benson Optical Co., Minneapolis, Minn. **Booths 43 and 44**

Every ophthalmologist will be interested in the newest items in the field of opticianry to be exhibited.

Blakiston Co., Philadelphia, Pa.
Booth 60

Warren Johnston and Donald Hicks, educational representatives in the Middle West, will display Blakiston's complete line of textbooks and clinical books. They will be particularly anxious to show you Doggart: **Ophthalmic Medicine**, Stevenson: **Recent Advances in Otolaryngology**, Hartridge: **Recent Advances in the Physiology of Vision, Proceedings of the First Clinical ACTH Conference**, and **Blakiston's New Gould Medical Dictionary.**

Boll & Lewis Optical Co., Chicago, Ill.
Booth 41

We will have on display a Kompact Refracting Unit, Tangent Screen, trial frames and hand diagnostic equipment, the modern trends in eye wear, as well as the latest developments in single vision and multifocal lenses.

Camel Cigarettes, New York, N. Y.
Booths 27 and 28

CAMEL Cigarettes will feature color slides of background data from their newest research. After weekly examinations of the throats of hundreds of men and women smoking CAMEL Cigarettes exclusively for thirty days, throat specialists reported "Not one single case of throat irritation due to smoking CAMELS."

Cameron Surgical Specialty Co., Chicago, Ill. **Booth 74**

See the New Anthony-Fisher Suction Coagulating Handle and Electrodes; cauteradios and cauterodynes for ocular, aural, nasal and oral electro-surgery, electro-cauterization and electro-coagulation; telescopic bronchoscopes, esophagoscopes and laryngoscopes; the new Boros Flexible Esophagoscope and Flexible Gastroscopes with treated and coated lenses; electro-diagnostic lamps and instrument sets; mirror-type headlights; binocular spectacle loupes and other specialties for ophthalmology and otolaryngology.

Canadian Radium & Uranium Corp., New York, N. Y. **Booth 59**

Radium D and E in the new truncated cone-shaped applicator will be shown, together with a new storage-carrying case. The improved storage-carrying container for the nasopharyngeal radium applicator will be demonstrated, together with a lead storage well for those who wish unusual protection from radiation.

Coca-Cola Co., Atlanta, Ga.
Booths 110 and 111

Ice-cold Coca-Cola served through the courtesy and co-operation of the Coca-

Cola Bottling Company of Chicago, Inc., and The Coca-Cola Company.

Coreco Research Corp., New York, N. Y.
Booth 14

The "CORECO" Automatic Color Camera is designed to photograph all surface areas of the body—from 1 to 1 close-up pictures to half-body size—and all cavities of the human body, such as mouth, throat, ear, nose, vagina, and rectum. The camera carries its own specially developed, fully color-corrected bulb and a mechanism for complete control of its color temperature and exposure within the camera itself. There is an automatic view finder synchronized with the automatic camera mechanism to permit viewing until a fraction of a second before exposure. The camera provides for automatic focusing.

An X-ray film applicator is included, making it possible to record all sizes and densities of X-ray film on kodachrome transparencies.

Davis & Geck, Inc., Brooklyn, N. Y.
Booth R-6

Davis & Geck, surgical suture manufacturers, will exhibit a complete line of absorbable and nonabsorbable suture materials and suture-needle combinations.

The D&G line of eye sutures includes a comprehensive group of suture-needle combinations especially developed for ophthalmic surgery. Literature describing the products will be available.

DeVilbiss Co., Toledo, Ohio
Booths 15 and 16

The DeVilbiss Company welcomes your visit to its exhibit of professional and prescription types of atomizers, nebulizers, and vaporizers. Among the newly developed products is the No. 33 Pocket Atomizer, designed to offer the advantages inherent in an uninterrupted treatment schedule and suitable for any solution. The No. 740 Hand Pump and Nebulizer, which makes a noteworthy contribution to the simplification of the home administration of aerosols, will be on display, as well as the new No. 149 Overnight Vaporizer.

Doho Chemical Corp., New York, N. Y.
Booth 32

The Doho Chemical Corporation, makers of AURALGAN and O-TOS-MO-SAN, are proud to announce their new nasal decongestant, RHINALGAN—a balanced formulation of two active chemical compounds that gives prolonged vasoconstriction—used as a spray, in our patented Dohony Spray-O-Mizer (combination dropper and spray). Pleasant tasting, with no systemic effect (pressor or respiratory), it can be safely used for infants and children.

Our representatives will be happy to explain the merits of RHINALGAN and distribute samples of this new innovation.

Encyclopaedia Britannica, Inc., Chicago, Ill.
Booth R-3

Grieshaber Swiss Eye Instrument Co., Chicago, Ill.
Booth 38

Exclusive United States and Canadian distributor for Ernst Grieshaber, Schaffhausen, Switzerland, ophthalmic instruments.

Our display will feature world famous Swiss ophthalmic cutting instruments, made to standards of greatest accuracy and known to promote highest efficiency and shorter time of healing.

Featured will be the widely acclaimed Castroviejo cataract needles and Castroviejo instruments.

We offer prompt deliveries from our large stock of Swiss ophthalmic instruments.

Grune & Stratton, Inc., New York, N. Y.
Booth 35

Among the new and standard books to be shown by Grune & Stratton, Inc., are **Principles of Ophthalmology** by T. Henderson, **Physiology of the Eye** by Arthur Linksz, **Occupational Eye Diseases** by Joseph Minton, **Acute Laryngotracheobronchitis** by A. H. Neffson, **Anatomy and Physiology of the Larynx** by V. E. Negus, **Clinical Audiology** by Maurice Saltzman, **Histology and Histopathology of the Eye** by I. G. Sommers, **Surgery of the Eye** by Meyer Wiener, **Speech Problems of Children** by Wendell Johnson, and **Language for the Preschool Deaf Child** by Grace Harris Lassman.

Copies of **The British Journal of Ophthalmology** and of **Ophthalmic Literature** will also be shown.

Guild of Prescription Opticians of America, Inc., New York, N. Y.
Booth 47

An illustrated presentation, with lantern slides, of "Sources of Patient Complaints Arising from Dispensing Procedures."

Paul B. Hoeber, Inc., New York, N. Y. Medical Book Department of Harper & Brothers **Booth 34**

At the Hoeber-Harper booth you will find a complete array of distinguished books. Among titles of particular value are the now complete set of Berliner's **Biomicroscopy of the Eye; Modern Trends in Ophthalmology**, edited by Arnold Sorsby; Gesell's **Vision**; Ritvo's **Roentgen Diagnosis of Diseases of the Skull**, and many others. Be sure to visit the Hoeber-Harper booth and see the entire list of Hoeber books in print.

Paul B. Hoeber will be in charge.

Hollister-Stier Laboratories, Wilkinsburg, Pa. **Booth 46**

The importance of allergy in the practice of otolaryngology is increasing and is evidenced by the fact that the specialty has its own allergy society. Hollister-Stier Laboratories are devoted exclusively to the preparation of allergenic materials and have worked with the American Society of Ophthalmologic and Otolaryngologic Allergy since its inception to prepare extracts which are useful in diagnosing and treating allergy cases.

Attendants at our booth will supply information on any of the products and services designed for your needs, including complete kits and individual items for office and laboratory.

Ion Optical Co., Detroit, Mich. Successors to Uhlemann Optical Company of Michigan **Booth R-12**

Keystone View Co., Meadville, Pa.
Booths 25 and 26

The Keystone View Company will exhibit its complete line of binocular testing and training instrumentation. Instruments and materials for the orthoptic handling of squint, amblyopia ex anopsia, suppressions, convergence and divergence anomalies, accommodation training and rapid perception will be shown.

Of special interest will be the new Home Training Services. These services, that have been widely used for many years, now have been made more valuable through a new stereoscope, new patient's manual, and a professional manual for the doctor.

Competent personnel will be available to demonstrate technics and instruments.

Kono Manufacturing Co., Woodside, N. Y.
Booth 109

Lea & Febiger, Philadelphia, Pa.
Booth 51

The Lea & Febiger exhibit will hold the interest of ophthalmologists and otolaryngologists because of their display of such outstanding books as Krimsky—**Binocular Imbalance**; Spaeth—**Principles and Practice of Ophthalmic Surgery**; Cowan—**Refraction of the Eye**; Gifford—**Ocular Therapeutics**; Kessler—**Principles and Practices of Rehabilitation**; Pullen—**Communicable Diseases**; Epstein—**Regional Dermatologic Diagnosis**; Holmes and Schulz — **Therapeutic Radiology**; Pohle—**Clinical Radiation Therapy**; Tuft—**Clinical Allergy**; Wiggers—**Physiology in Health and Disease**; Ballenger—**Diseases of the Nose, Throat and Ear**; and many others.

J. B. Lippincott Co., Philadelphia, Pa.
Booth R-8

J. B. Lippincott Company presents an interesting and active exhibit of professional publishing. With the "pulse of practice" centering in an advisory editorial board of active clinicians who constantly review the field, current and coming trends in medicine and surgery are known continually. On the studied recommendations of these medical leaders, Lippincott Selected Professional Books are undertaken.

Mager & Gougelman, Inc., Chicago, Ill.
Booths 19½ and 20

Mager & Gougelman, Inc.'s technical exhibit will consist of the finest display of artificial eyes in both glass and plastic. The display will include all types of motility implants, sphere implants, radiation therapy eye shields, intraocular foreign body locators, drains and conformers. A film will be shown at all times on the latest surgical technics for enucleation with motility implants. The display will feature facial restorations and orbital appliances.

Maico Co., Inc., Minneapolis, Minn.
Booths 66 and 67

This year the Maico exhibit will feature an entirely new line. The improved E-2 Research Audiometer, the new H-1 Professional Model Audiometer, the new F-1 Portable School Audiometer, together with Maico's sensational new unit, the Train-Ear, for speech teaching and aural rehabilitation, will make this exhibit one of extreme interest and well worth your time. Also to be shown will be the new Model J Hearing Aid which combines extremely small size with fidelity of tone, range of amplification and flexibility of adjustment to a degree which Maico has not previously been able to equal.

Marcelle Cosmetics, Chicago, Ill.
Booth 54

New developments and new products in the Marcelle hypo-allergenic Cosmetic line will be demonstrated at the Marcelle exhibit. Representatives will include members of the Marcelle research organization who will give data and information on the role of Marcelle hypo-allergenic Cosmetics in the physician's practice.

Medical Bureau, Chicago, Ill.
Booth R-9

Burneice Larson offers the facilities of The Medical Bureau, an organization serving as counselor in problems of medical personnel to medical schools and universities, public health organizations, hospitals and institutions, group clinics, physicians in private practice and industry.

The records of diplomates of the American Boards qualified to head their departments, younger physicians interested in obtaining training and, also, administrators, executive and supervising nurses, scientists, laboratory technicians, x-ray technicians, social workers and dietitians are available to those interested in the completion or reorganization of their staffs.

Medical Case History Bureau, New York, N. Y. **Booth 112**

Medical Case History Bureau suggests: Be a modern minute man who records his patients' minutes in fewer minutes with the hours-saving INFO-DEX System.

Your present history charts easily incorporated, without rewriting, into streamlined new system which has special features like cross-index for quick reference, cotter pin for ever-hold, never-lose charts.

Many physicians consider concise INFO-DEX charts a MUST.

Medical Film Guild, New York, N. Y.
Booth A

"Medical Films That Teach" presents a refresher course in fundamental medical problems. Each film subject is produced in the manner of a textbook, profusely illustrated, offering information comparable to that found in postgraduate courses as presented at our leading medical schools. These films review such subjects as Allergy-Immunology Diagnosis and Treatment, A Clinic on Deafness, A Clinic on Sigmoid Sinus Thrombosis, Function of the Ear in Health and Disease, Anatomy of the Ear, Chronic Purulent Otitis Media, A Clinic on Petrositis with Meningitis, A Clinic on Acute Mastoiditis, Otitis Media in Pediatrics, Otoscopy in the Inflammations, Hypothyroidism: Etiology-Diagnosis-Treatment, Management of the Failing Heart, Arterial Blood Pressure Technic-Physiology Measurement, Cervicitis: Etiology and Treatment, Bronchial Catheterization and Bronchopulmonary Lesions, Bronchial Asthma, Treatment of the Major Neuralgias, Treatment of Urinary Infections, Rheumatoid Arthritis and many others. These films are available at no charge to intern groups, hospital staff conferences, limited nurses' groups and to general medical meetings. This includes projection service at no charge and is arranged through grants for postgraduate instruction.

E. B. Meyrowitz Surgical Instruments Co., Inc., New York, N. Y., Booths 79 and 80

E. B. Meyrowitz Surgical Instruments Co., Inc., this year celebrating its seventy-fifth anniversary, specializes in eye, ear, nose and throat instruments. Displayed will be a complete assortment of the above-mentioned instruments, together with tonometers, perimeters, et cetera. New needle holders, forceps and scissors will be of especial interest to the ophthalmic surgeon.

Microtone Co., St. Paul, Minn.
Booth 78

The Microtone Company A.D.C. Audiometer exhibit will feature the new Microtone A.D.C. Clinical Pure Tone Speech Audiometer. This outstanding in-

strument is available in console or portable models with provisions for additional amplifiers and accessories. Two-room testing is possible with this new, compact unit as well as standard tests given in the average otologist's office. Your inspection will be cordially welcomed.

Modern Optics, Inc., Houston, Texas
Booth R-2

Modern Optics, Inc., Houston, Texas, will show at its exhibit a radical innovation in lenses for the postoperative aphakic patient. These "cataract lenses" will be designed for each individual patient, and they will always be more costly than the usual lens. Virgil Hancock, president of Modern Optics, on behalf of the Company invites your comment both as to the new lens itself and as to the possible market for such a high-priced item.

C. V. Mosby Co., St. Louis, Mo.
Booth 86

You are invited to visit the C. V. Mosby Company booth, where you will find many new and interesting titles. Information will be available on Adler, **Physiology of the Eye**; Allen, **Strabismus**; Meakins, **Practice of Medicine**; new edition of Tassman, **Eye Manifestations of Internal Diseases.** You can see and examine full volumes of Kuhn, **Eyes and Industry**; Sorsby, **Genetics in Ophthalmology** and Sorsby, **Systemic Ophthalmology.**

V. Mueller & Co., Chicago, Ill.
Booths 1 thru 6

The new Nugent Vacuum Unit for cataract extraction and several other improved erisophakes will be features of the V. Mueller and Company exhibit. Shambaugh instruments for fenestration, including the Shambaugh Operating Microscope, and the latest instruments for rhinoplasty will be among other interesting items in this comprehensive display of fine new and standard instruments for all surgery of the eye, ear, nose and throat.

New Jersey Optical Co., Irvington, N. J.
Booth 48

The exhibit of the New Jersey Optical Company will be headed by Mr. Sydney Freedman, vice-president.

He reports that in addition to their Duke and Duchess, which are 1/10 12 K. Gold Filled throughout, they will feature a radically styled spectacle in this new classification.

Novocol Chemical Mfg. Co., Inc., Brooklyn, N. Y.
Booth 68

A cordial invitation is extended to all to visit with us. On display will be the popular aspirating-type Anestube syringe which is daily gaining new friends in the specialties of ophthalmology, otorhinolaryngology and rhinoplasty. For the safety and comfort of your patients you should learn more about MONOCAINE HCl solutions. A qualified representative will be in attendance.

Obrig Laboratories, Inc., New York, N. Y.
Booth 102

The Obrig Laboratories exhibit will consist of:

1. The original molded plastic contact lens
2. The solutionless molded Obrig "Lacrilens" contact lens
3. The Obrig corneal contact lens

Of added interest will be a unique collection of contact lenses made by various manufacturers in America, Europe and South America during the past fifty years.

Paravox, Inc., Cleveland, Ohio
Booth R-1

Paravox, Inc., manufacturer of Paravox Hearing Aids, will again be among the technical exhibitors at the forthcoming American Academy of Ophthalmology and Otolaryngology convention. The Paravox exhibit will include a beautiful plastic display, showing the various component parts of the Paravox YM Model Hearing Aid in the proper sequence of manufacture, and how each of the parts finds its way into the final assembly, forming the completed unit. This display is attractively illuminated by neon tubes.

Philip Morris & Co. Ltd., Inc., New York, N. Y. **Booth 75**

Philip Morris and Company will show the results of research on the irritant effects of cigarette smoke. These results show conclusively that Philip Morris cigarettes are less irritating than other cigarettes. An interesting demonstration will be made on smokers at the exhibit which will show the difference in cigarettes.

Geo. P. Pilling & Son Co., Philadelphia, Pa. Booths 63 thru 65

The George P. Pilling & Son Company will again bring a complete line of eye, ear, nose, throat and broncho-esophagoscopic instruments. Several new styles of Anterior Commissure Laryngoscopes will be featured, including the new improved Sam Roberts Self-Retaining Laryngoscope.

Visit the Pilling Exhibit to see the new modern Pilling Portable Storage Battery Unit.

Professional Press, Inc., Chicago, Ill. Booth 50

The Eye, Ear, Nose and Throat Monthly is now in its twenty-ninth year of publication. It is intended as a practical journal for the ophthalmologist and the otolaryngologist. Its contents are well departmentalized, rendering it a simple matter to refer to any special section desired. The original articles are contributed by outstanding authors and a fair balance of material is maintained in the eye and ear, nose and throat sections. Sample copies will be available to members of the Academy.

Radium Chemical Co., Inc., New York, N. Y. Booths 30 and 31

Radium Chemical Company, Inc., will display the Monel metal radium applicator used in the treatment of hyperplastic lymphoid tissue in the nasopharynx. Radium Chemical Company was the originator of this applicator and has supplied more than 1400 to otolaryngologists in the United States.

Beta ray eye applicators and radon equipment will also be displayed.

Ritter Co., Rochester, N. Y. Booths 69 thru 73

Unit and Motor Driven Chair equipment for the ear, nose and throat specialists. Motor Driven Tables with or without Mobile and Explosion-proof bases.

W. B. Saunders Co., Philadelphia, Pa. Booth 55

We invite all doctors attending the meeting of the American Academy of Ophthalmology and Otolaryngology to visit our exhibit, where we will display a complete line of our books including Hyman's **Integrated Practice of Medicine,** Bockus' **Postgraduate Gastro-enterology,** Wells' **Clinical Pathology,** Berens' **Diseases of the Eye,** Gifford-Adler's **Ophthalmology,** Mitchell and Nelson's **Pediatrics,** Boies' **Otolaryngology,** Jackson and Jackson's **Broncho-Esophagology,** and many other new books and new editions.

Schering Corp., Bloomfield, N. J. Booth 33

Sodium Sulfacetimide in the form of ophthalmic solution and ophthalmic ointment, as well as nasal solution, will be featured at the Schering exhibit. Graminasin nasal solution containing gramicidin will also be highlighted.

Schering professional service representatives will be present to welcome your inquiries on other Schering pharmaceutical specialties.

Shuron Optical Co., Inc., Geneva, N. Y. Booths 81 and 82

Shuron will feature the Optical Wardrobe idea of providing patients with eyewear styled for different occasions, such as Browline for everyday wear, Shurset for "dress up" wear and Shelltex for casual and sports wear. A wide assortment of modern styles will be on display.

Smith, Kline & French Laboratories, Philadelphia, Pa. Booth 45

DEXAMYL—S.K.F.'s balanced combination of 'Dexedrine' Sulfate (dextroamphetamine sulfate, S.K.F.) and 'Amytal' (Amobarbital, Lilly)—is a new preparation of wide usefulness in everyday practice. Combined in DEXAMYL, 'Dexedrine' and 'Amytal' work together to provide symptomatic relief from mental and emotional distress.

Soft-Lite Lens Co., Inc., New York, N. Y. Booths 104 and 105

Sonotone Corp., Elmsford, N. Y. Booth 83

Sonotone Corporation, manufacturers and distributors of hearing aids and audiometers, this year presents a new, portable audiometer for doctors. Also on display will be the Model 30 Screening Audiometer, available for either battery or power-line operation. Sonotone's complete line of four tiny self-contained hearing aids will also be exhibited, as well as the line-operated Model 50 Professional Desk Set, designed for use in doctors' offices and for auditory training.

C. M. Sorensen Co., Inc., New York, N. Y. Booth 42

The C. M. Sorensen Company is exhibiting various suction and pressure units; ear, nose and throat treatment units; hydraulic chairs; central suction and pressure installations.

Storz Instrument Co., St. Louis, Mo. Booths 99, 100 and 101

Storz Instrument Company will display eye, ear, nose and throat and plastic surgery instruments and also the Haag Streit Slit Lamp. We will have a complete assortment of standard models and shall be showing many new items and current modifications of standard patterns.

Surgical Mechanical Research, Los Angeles, Calif. Booths 93 and 94

We will exhibit equipment advertised in this issue of the TRANSACTIONS, as well as our new electrically heated wash bottle, physician's grip and numerous small items for the office.

Charles C Thomas, Springfield, Ill. Booth 49

Featured in this display: Callahan's **Surgery of the Eye: Injuries**; Cogan's **Neurology of the Ocular Muscles**; Holmes' **The Nose**; Bast and Anson's **Temporal Bone and the Ear**; Lyle's **Neuro-Ophthalmology**; Canfield's **Audiology**; and Mykelbust's **Your Deaf Child.** Orders are being taken for Dr. Walter B. Lancaster's new book, **Refraction and Motility.** Several new monographs are being added to **American Lectures in Otolaryngology, Audiology and Broncho-Esophagology** edited by Dr. Norton Canfield. All books are arranged for your comfort and convenience.

Titmus Optical Co., Inc., Petersburg, Va. Booth 56

The new Titmus Corrected Curve Series of Single Vision Lenses will be featured by the Titmus Optical Company, Inc., at its convention booth. The new Titmus Corrected Curve Series, accurately computed for all powers, eliminates the effect of marginal astigmatism within the most used ranges. This practical, all-purpose ophthalmic lens is equally effective for general and for detailed vision. Members of the Titmus research staff will be on hand to demonstrate the new corrected curve series.

Uhlemann Optical Co., Chicago, Ill. Booths 52 and 53

We invite you to see our outstanding display of "glasses by Uhlemann" . . . a collection of styles with technical qualities. Our exhibit will include new features in ophthalmic equipment.

Univis Lens Co., Dayton, Ohio Booths 39 and 40

The Univis Lens Company will feature its newest diagnostic tool, the Presbyopic Trial Set. Successor to the Univis Trifocal Trial Set, introduced last year, the new tool represents significant advance in presbyopic prescription aids; effectively depicting comparative characteristics of trifocal and bifocal application.

Victory Optical Manufacturing Co., Newark, N. J. Booth 108

Victory Optical Manufacturing Company will exhibit its complete line of zyl frames, along with its latest presentations —the V-SET line. A new display, artistically styled, is being constructed especially for this event.

Mr. Vincent Salierno, president of the company, states that special emphasis will be placed on the new "Rosalie" frame, recently introduced, and on the new zyl frames in metallic-color effects of gold, grey, blue and red, the royal and deluxe gold, as well as the popular Honey, Doll and Commentator frames.

Among the Victory representatives who will attend the convention are Vincent Salierno, Joseph G. Bianco and Harold Abrams.

Welch-Allyn Co., Auburn, N. Y. Booth 17

The new Sandura Diagnostic Instrument Case features Welch-Allyn's display which includes the Lancaster Red-Green Test, ophthalmoscopes, retinoscopes, otoscopes, illuminated nasal dilatators, laryngoscopes, and nasopharyngoscopes.

White-Haines Optical Co., Columbus, Ohio Booths 21 and 22

White Laboratories, Inc., Newark, N. J. Booth 103

White Laboratories, Inc., will have an interesting and unusual display—the latest products of White's research. Courteous medical service representatives in attendance will appreciate the opportuni-

ty to discuss with you the clinical background and therapeutic merit of these and other White's products. You are cordially invited to visit this booth.

Williams & Wilkins Co., Baltimore, Md.
Booth 36

Among new books displayed will be **Tumors of the Head and Neck,** by Ward and Hendrick; **Physiological Basis for Medical Practice,** new fifth edition, by Best and Taylor; **Nasal Sinuses,** new second edition, by O. E. Van Alyea; **Hearing Tests and Hearing Instruments,** by Watson and Tolan. There will also be such standard works as: **Histopathology of Ear, Nose, and Throat,** by Eggston and Wolff; **Neuro-Ophthalmology,** by Walsh.

Winthrop-Stearns, Inc., New York, N. Y.
Booth R-7

The members and guests of the American Academy of Ophthalmology and Otolaryngology are cordially invited to visit the Winthrop-Stearns booth, where representatives will be on hand for your service. Featured products are Sulfamylon and Sulfamylon with Streptomycin for effective topical chemotherapy of eye, ear, nose and throat infections; Neo-Synephrine Thenfadil, a combination of the time-tested, dependable decongestant, Neo-Synephrine, with a new, potent, well tolerated antihistaminic, Thenfadil; Pontocaine, reliable topical anesthetic with prolonged action and good penetration.

Max Wocher & Son Co., Cincinnati, Ohio
Booths 76 and 77

The Max Wocher & Son Company will have a complete display of the latest in instruments and surgical supplies for ophthalmology and otolaryngology. This year Wocher's will be featuring many imported items not available for some time.

Wottring Instrument Co., Columbus, Ohio
Booths 23 and 24

The Wottring Instrument Company will have a complete line of orthoptic equipment on display. The exhibit will consist of the Troposcope, Royal Rotoscope, DeLuxe Rotoscope, Owens Devicmeter, and the electric table.

There will also be on exhibit a complete line of orthoptic accessories and targets. Among the various sets of targets will be the new Walraven ARC targets.

Zenith Radio Corp., Chicago, Ill.
Booths 84 and 85

The Zenith Radio Corporation will exhibit the latest air and bone conduction hearing aids produced by this manufacturer of quality radios and television sets. Zenith representatives will be pleased to explain how any physician can test one of these instruments for 30 days without cost or obligation to him or his hard-of-hearing patients.

Zylo Ware Corp., Long Island City, N. Y.
Booth R-5

We will display a comprehensive line of the quality Zylonite frames that we manufacture. Special emphasis will be laid upon our Hi-Mount frame, which is the latest innovation and advance in frame design

This frame is ingenious in its construction; lenses are held in a light Zylonite rim, which is very inconspicuous and which we are apt to refer to as invisible.

There are no screws, washers or other metal parts to contend with in the mount of lenses, which is of course a great boon to the laboratory technician. In addition, we will be showing our complete line of Zylonite frames that are meeting with increasing favor among the profession.

News - - - Notes

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations: October 3-6, Chicago, Ill., at the Palmer House; and January 8-11, New York City, at the Hotel Biltmore.

Officers of the Board are: president, Dr. Arthur W. Proetz, St. Louis, Mo.; Vice-president, Dr. LeRoy A. Schall, Boston, Mass.; and secretary-treasurer, Dr. Dean M. Lierle, Iowa City, Iowa.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

The annual meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy will be held at the Palmer House in Chicago on Saturday, Oct. 7, 1950.

At the morning session a symposium on "The Use of Anti-Histaminics in the Common Cold" will be presented. Composing the symposium will be Dr. Arthur Proetz, "The Physiology of the Nose in Relation to the Common Cold and Anti-Histaminics"; Capt. John M. Brewster, "The Use of Anti-Histaminics in the Treatment of the Common Cold"; Dr. Charles H. Ramelkamp, Jr., "The Epidemiology of the Common Cold and Its Treatment with Anti-Histaminics"; Dr. French K. Hansel, "The Differentiation of the Common Cold from the Allergic Reaction Resembling the Common Cold."

During the afternoon Dr. Anderson Hilding will speak on "The Bronchi in Asthma"; Dr. Paul Holinger will discuss "The Use of Bronchoscopy in the Treatment of Bronchial Asthma"; Dr. Conrad Berens will read a paper on "The Relation of Allergy to Ophthalmology"; and Dr. Samuel A. Hanser will present "Recent Studies in Ocular Allergy."

The annual dinner of the Society will be held at the Palmer House on Thursday evening, October 12. Dr. Herbert Rinkel will be guest of honor. Immediately following the dinner the Society will hold a joint session with the American Academy of Ophthalmology and Otolaryngology. (See p. 772)

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

The offices of the International Association for Prevention of Blindness have been transferred to the offices of the President, Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France.

PAN-AMERICAN CONGRESS ON OPHTHALMOLOGY

The Secretary-General of the Fourth Pan-American Congress on Ophthalmology, Dr. Luis Sanchez Bulnes, has announced that the Congress will meet in Mexico City Jan. 6-12, 1952.

CALIFORNIA

The University of California Medical School has announced that it will present a Postgraduate Course in Ophthalmology at Toland Hall, University Hospital, September 11-16. Dr. Frederick C. Cordes is general chairman, and the officers of instruction are all members of the faculty of the University of California Medical School. On Friday evening, September 15, Dr. Algernon B. Reese, clinical professor of ophthalmology, Columbia University, has been invited to deliver the fifth annual Francis I. Proctor Lecture on Ophthalmology. The subject of his lecture will be "Retrolental Fibroplasia."

Application for enrollment in the course should be made to Dr. Stacy R. Mettier, head of postgraduate instruction, Medical Extension, University of California Medical Center, San Francisco 22, Calif.

DISTRICT OF COLUMBIA

A nine month course for the training of orthoptic technicians will begin on Oct. 2, 1950, under the auspices of the George Washington University School of Medicine. Application should be made to Dr. Ronald A. Cox, Department of Ophthalmology, George Washington Hospital, 901 Twenty-third Street, N.W., Washington, D. C.

ILLINOIS

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Abraham

Lincoln Hotel in Springfield, Ill., September 22-24. The guest speakers will be Dr. Girolamo Bonaccolto of New York, who will give several lectures on ophthalmology, and Dr. Bayard T. Horton of Rochester, Minn., who will speak on "Histaminic Cephalalgia" and "The Treatment of Retrobulbar Neuritis with Histamine." Drs. C. L. Pannabecker and A. L. Paisley, members of the Society, will also speak.

George W. Wallerich, president of V. Mueller & Company, Chicago, died July 3 after a long illness.

KANSAS

Dr. G. O'Neil Proud, instructor in the department of otolaryngology at Washington University School of Medicine, St. Louis, Mo., has been appointed chairman of the department of otolaryngology at the University of Kansas School of Medicine, Kansas City, effective July 1. Dr. Sam E. Roberts, chairman of the department since 1928, who has been made chairman emeritus, will devote his time to private practice. Dr. Proud received his medical degree from Washington University in 1939 and has served on the staffs of St. Louis Children's and Barnes hospitals. He is a lieutenant in the U. S. Naval Reserve.

PENNSYLVANIA

The third annual clinical conference of the Wills Eye Hospital, Philadelphia, will be held at the hospital March 16-17, 1951. As in other years, papers which reveal the latest developments in ophthalmic treatment and procedures will be presented by many of the nation's outstanding ophthalmologists.

The Western Pennsylvania Eye, Ear, Nose and Throat Society held its thirty-third meeting in Indiana, Pa., on May 25. Dr. John Dickinson of Pittsburgh gave a paper on "Broader Aspects of Otology," and Dr. Robert Rohm of Pittsburgh spoke on "The Management of Strabismus."

NEW YORK

The Eastern New York Eye, Ear, Nose and Throat Association will hold its Annual Clinic Day on October 5 at the Albany Hospital outpatient department. Dr. John Cetner is chairman of the program committee and an interesting program is being planned.

POSITIONS AVAILABLE

Ophthalmologist: A full-time teaching fellowship for an ophthalmologist who is certified or eligible for certification, is available at the Manhattan Eye, Ear and Throat Hospital. In addition to teaching duties, there would be opportunity to work in research laboratories and the Eye Bank. For further information write to Mr. Fred Heffinger, Superintendent, Manhattan Eye, Ear and Throat Hospital, 210 East Sixty-Fourth Street, New York 21, N. Y.

The Department of Otolaryngology and Oral Surgery at the University of Iowa Medical School will have four residencies available July 1, 1951. Applications should be submitted by November 1, 1950, as the appointments are made December 1, 1950. If you are interested, please write the Department of Otolaryngology and Oral Surgery, University Hospitals, Iowa City, Iowa, for information.

Wanted: The community of Crossville, Tenn., has great need for the services of a physician specialist in eye, ear, nose and throat work. Crossville is located on the Cumberland Plateau at an elevation of 2000 feet, has a population of 3500, with a trade and service area containing approximately 30,000 people. The area is largely agricultural, with some light manufacturing industry. There is a high percentage of individual farm and home ownership. The hospital is a new one of 50 beds and 12 bassinets, city owned and operated. For further information write to Mr. M. B. Shroyer, Administrator, Cumberland Medical Center, Crossville, Tenn.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS
President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Dr. A. Churchill, 66 Boulevard Saint-Michel, Paris, 6, France

INTERNATIONAL COUNCIL OF OPHTHALMOLOGY
President: Prof. F. W. Nordenson
Secretary: Dr. Halger Ehlers, Rigshospital, Copenhagen, Denmark

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA
President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY
President: Dr. Conrad Berens
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: IV Pan-American Congress of Ophthalmology early in 1952

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY, PUERTO RICO CHAPTER
President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY
President: Prof. Justo Alonso
Secretary: Dr. Chevalier L. Jackson, 255 South 17th Street, Philadelphia, Pa.

FOREIGN

ALL-INDIA OPHTHALMOLOGICAL SOCIETY
President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

AUSTRIAN OTOLARYNGOLOGICAL SOCIETY, VIENNA
President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time and Place: Annually. September 18-20, 1950. Bad Gastein, Austria

BELGIAN OPHTHALMOLOGIC SOCIETY
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION
Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS
President: Mr. H. V. Forster
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

CHENGTU OPHTHALMOLOGICAL SOCIETY
President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

CHINESE OPHTHALMOLOGY SOCIETY
President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING
President: Dr. H. T. Pi
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping
Time and Place: Last Friday of each month. Peiping Union Medical College

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG
President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik
Time and Place: September 18-20, 1950. University of Munich

FACULTY OF OPHTHALMOLOGISTS
President: Dr. Frank W. Law, 45 Lincoln's Inn Fields, London, W.C. 2, England
Secretary: Dr. J. H. Doggart, F.R.C.S.

GERMAN OPHTHALMOLOGICAL SOCIETY
President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: I. Csapody
Secretary: E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

ISRAEL OPHTHALMOLOGICAL SOCIETY
President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP
President: Prof. A. W. Mulock Houwer
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Mr. J. S. Arkle
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle, Liverpool, Sheffield and Bradford

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. D. M. MacRae
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY OF ALBERTA
President: Dr. A. Fettes
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: September 1950, Calgary, Alberta

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA
President: Dr. Arthur H. Joyce
Secretary: Dr. Arnold L. Lance, 135 Macquarie Street, Sydney, New South Wales

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN
President: Prof. Dr. Holger Ehlers
Secretary: Dr. Carl Johan Moellenbach, Rigshospitalets oejenafdeling, Copenhagen O, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT
President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mahmoud Lutfi, Ophthalmic Hospital, Giza
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah, 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ
Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Neustra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND
President: Dr. L. S. Talbot
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA
President: Dr. M. Fine
Secretary: Dr. R. L. H. Townsend, National Mutual Building, Church Square, Cape Town

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM
President: Mr. M. H. Whiting
Honorary Secretaries: Mr. A. G. Cross and Mr. A. J. B. Goldsmith, 45 Lincoln's Inn Fields, London W. C. 2, England
Time and Place: 1951 Congress of the Society, March 29-31, London

OXFORD OPHTHALMOLOGICAL CONGRESS
Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury
Time and Place: No meeting in 1950 due to International Meeting in London in June.
Next meeting first week of July 1951, Oxford

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Geminiano de Ocampo
Secretary-Treasurer: Dr. Carlos V. Yambao, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY
President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY
President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY
President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB
President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Claremont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA
President: Dr. Diego M. Arguello
Secretary: Dr. Pedro F. Garcia Nocito, Vicente Lopez 1756 A, Buenos Aires, Argentina
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA

President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile
Place: Santiago

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORRINOLARINGOLOGIA

President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA

President: Prof. Lorenzo Comas
Secretary: Dr. Heriberto Buch Granados, Calle B No. 668 ent. 27 y 29, Vedado, Habana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA

President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA

Chairman: Dr. Roberto Obregon Oliva
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA

President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL

President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA

President: Dr. Natalicio de Farias
Secretary: Dr. Ismar Pereira, Praca Floriano 55, 5° andar, Rio de Janeiro, Brazil
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DEL NORTE

President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS

President: Prof. Hilton Rocha
Secretary: Dr. Oswaldo Carvalho
Place: Belo Horizonte, Minas Gerais, Brazil

SOCIEDADE DE OFTALMOLOGIA E OTORINOLARINGOLOGIA DE RIO GRANDE DO SUL

President: Dr. Luiz Assumpcao Osorio
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO

President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE FRANCAISE D'OPHTHALMOLOGIE

Secretary-General: Dr. Edward Hartmann, Hospital Lariboisiere, Paris 10, France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm, Sweden

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: R. Lang, M.R.C.S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England
Time and Place: September 23, 1950, Portsmouth and Southern Counties Eye and Ear Hospital, Southsea
October 21, 1950, Royal Surrey County Hospital, Guildford

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. Mackenzie Brown
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 8-13, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

President: Dr. Kenneth L. Craft
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Ralph A. Fenton
Secretary: Dr. Louis H. Clerf, 1530 Locust, Philadelphia 2, Pa.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.
President: Dr. Robert C. Martin
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
EASTERN SECTION
Vice-President: Dr. Robert L. Moorhead
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
MIDDLE SECTION
Vice-President: Dr. J. Marion Sutherland
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.
SOUTHERN SECTION:
Vice-President: Dr. William D. Stinson
Secretary: Dr. Lewie M. Griffith, 301 Haywood Building, Asheville, N. C.
WESTERN SECTION:
Vice-President: Dr. Jesse B. Naftzger
Secretary: Dr. Russell M. Decker, 65 North Madison Avenue, Pasadena, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. A. Ray Irvine
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY
Chairman: Dr. William H. Johnston
Secretary: Dr. James M. Robb, 641 David Whitney Building, Detroit, Mich.

AMERICAN OPHTHALMOLOGICAL SOCIETY
President: Dr. Bernard Samuels
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 West 59th Street, New York 19, N. Y.

AMERICAN OTOLOGICAL SOCIETY
President: Dr. Philip E. Meltzer
Secretary: Dr. Gordon D. Hoople, Medical Arts Building, Syracuse 3, New York

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY
President: Dr. George E. Shambaugh, Jr.
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Building, Pittsburgh, Pa.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.
Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ontario

CANADIAN OPHTHALMOLOGICAL SOCIETY
President: Dr. R. F. Nicholls
Secretary: Dr. J. F. A. Johnston, 174 St. George Street, Toronto, Ontario

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. W. Griffin Jones
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, October through May. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
Chairman: Dr. Robert T. Wong
Secretary: Dr. Ogden D. Pinkerton, Suite 7, Young Building, Honolulu 13, T. H.
Time and Place: Third Thursday of each month (dinner meetings). Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. R. O. Porter
Secretary: Dr. Harry O. Frazier, 707 Medical Arts Building, Salt Lake City 1, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. William A. Wagner
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett L. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Robert L. Goodale
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. David R. Higbee
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter Street, San Francisco, Calif.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. V. E. Cortopassi
Secretary: Dr. A. J. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Alston Callahan
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: November 13-16. St. Louis, Mo.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Leonard Swigert
Secretary: Dr. James C. Strong, 227 Sixteenth Street, Denver, Colo.
Time and Place: Every third Saturday. Colorado Medical Center

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. Guy W. Smith
Secretary: Dr. Terry J. Gromer, 110 Metropolitan Building, Denver, Colo.
Time and Place: 6:30 p.m., first Saturday evening of each month. Oxford Hotel, Denver

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. Jerome Knauer
Secretary-Treasurer: Dr. Charles C. Grace, 145 King Street, St. Augustine, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Lester A. Brown
Secretary: Dr. Braswell E. Collins, 701 Elizabeth Street, Waycross, Ga.
Time and Place: First Friday and Saturday of March General Oglethorpe Hotel, Savannah, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. W. Dyar
Secretary-Treasurer: Dr. M. S. Harding, 308 Hume Mansur Building, Indianapolis, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa
Time and Place: 1950, Des Moines

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Malcolm C. Pfunder
Secretary-Treasurer: Dr. Frank Adair, 822 Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. W. L. Forster
Secretary-Treasurer: Dr. F. D. Hurd, Medical Arts Building, Great Falls, Mont.
Time and Place: Semi-annually. Summer, Bozeman; mid-winter, Diamond S Ranchotel, Boulder

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. G. M. Billings
Secretary: Dr. MacLean B. Leath, 529 North Main Street, High Point, N. C.
Time and Place: Annually, beginning second Monday in September. 1950 meeting, September 11-13, Hendersonville, N. C., with the South Carolina Eye, Ear, Nose and Throat Society.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Max Simons
Secretary: Dr. Richard S. Fixott, 1020 Southwest Taylor Street, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Old Heathman Hotel, Portland

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Jay G. Linn
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William M. Carpenter
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. C.
Time and Place: September 11-13. Hendersonville, N. C. (jointly with North Carolina Eye, Ear, Nose and Throat Society)

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Sam H. Sanders
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. V. R. Hurst
Secretary: Dr. John L. Matthews, 414 Navarro Street, San Antonio 5, Texas
Time and Place: December 1950. Dallas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Francis H. McGovern
Secretary-Treasurer: Dr. Peter N. Pastore, Box 25, Medical College of Virginia Station, Richmond 19, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Garnett P. Morison
Secretary: Dr. Melvin W. McGehee, 425 Eleventh Street, Huntington 1, W. Va.
Time: Biannual

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Anthony M. Sellitto
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: Second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January March, May and November. Akron City Club

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Lester A. Brown
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION
President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Mortimer A. Lasky
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Arthur L. Bennett
Secretary: Dr. Herbert R. Reitz, 446 Linwood Avenue, Buffalo, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Harold R. Watkins
Secretary-Treasurer: Dr. Philip R. McGrath, 843 Jefferson Building, Peoria, Ill.
Time and Place: September 22-24, Abraham Lincoln Hotel, Springfield

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Cecil B. Hert
Secretary-Treasurer: Dr. James L. McGraw, 619 University Building, Syracuse, N. Y.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. G. Dunn
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.
Time and Place: September 9-10. Sturgeon Bay, Wis.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Oliver E. Van Alyea
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. Derrick Vail
Secretary-Treasurer: Dr. J. Robert Fitzgerald, 3215 W. North Avenue, Chicago
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CLEVELAND OPHTHALMOLOGICAL CLUB
President: Dr. G. Leslie Miller
Secretary-Treasurer: Dr. Webb P. Chamberlain, Jr., 7405 Detroit Avenue, Cleveland, Ohio
Time and Place: Second Tuesday of November, January, February and April. Statler Hotel, Cleveland

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Wilfred E. Fry
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL SOCIETY
President: Dr. Cecil Lepard
Secretary: Dr. Arthur P. Wilkinson, 974 Fisher Building, Detroit 2, Michigan
Time and Place: Second Monday of each month, November through April. Wayne County Medical Society

DETROIT OTO-LARYNGOLOGICAL SOCIETY
President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. Byron H. Porter
Secretary-Treasurer: Dr. E. Martin Freund, 762 Madison Avenue, Albany 3, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. W. H. McKenzie
Secretary: Dr. C. Keith Barnes, 921 Neil P. Anderson Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. All Saints' Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Alan L. Sparks
Secretary-Treasurer: Dr. J. Lawrence Sims, 809 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. John McLeod
Secretary: Dr. James W. May, 1016 Rialto Building, Kansas City, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June. Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Alden H. Miller
Secretary: Dr. Victor Goodhill, 2007 Wilshire Boulevard, Los Angeles 5, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY
President: Dr. Victor Alfaro
Secretary: Dr. Frasier Williams, 1228 North Irving Street, Arlington, Va.
Time and Place: 7:00 p.m., third Tuesday in October, November, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. J. P. Wild
Secretary-Treasurer: Dr. Howard High, 324 East Wisconsin Avenue, Milwaukee 2, Wis.
Time and Place: 6:30 p.m., fourth Tuesday of each month, October to May. Athletic Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. Jules Brahy
Secretary-Treasurer: Dr. John V. V. Nicholls, 1414 Drummond St., Montreal, Quebec, Canada
Time: Second Thursday of October, December, February and April

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Milton L. Berliner
Secretary: Dr. John M. McLean, 525 East 68th Street, New York 21, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Samuel Gartner
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. Dusty's, 1739 South Brownlee Street, Corpus Christi, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly. Pittsburgh

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. Frank H. Wanamaker
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. William J. Hertz
Secretary: Dr. Paul C. Craig, 232 North Fifth Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. E. W. Perkins
Secretary-Treasurer: Dr. J. Warren Montague, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tetlie, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

ST. LOUIS OPHTHALMIC SOCIETY
President: Dr. Lawrence T. Post
Secretary: Dr. James H. Bryan, 330-332 Beaumont Medical Building, St. Louis 8, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB
President: Dr. Theodore Holstein, 523 Medico-Dental Building, Sacramento 14, Calif.
Secretary: Dr. John Berg
Time: Every third Thursday, eight months of the year

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
Chairman: Dr. A. Lloyd Morgan
Secretary: Dr. Joseph C. Hill, 174 St. George Street, Toronto 5, Ontario, Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WASHINGTON, D. C. OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Thomas Schnebly
Secretary-Treasurer: Dr. E. S. Caldemeyer, 1826 R Street, N.W., Washington 9, D. C.
Time and Place: Bimonthly, October through May. Kennedy-Warren Hotel

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. E. Imbrie
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of May and October

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY
President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

As of August 1, 1950

Aurex Model F ..... JAMA 138:294 (Sept. 25) 1948
Aurex Model H ..... JAMA 136:1099 (April 24) 1948

MFR. AUREX CORP.
1117 N. Franklin St., Chicago 10, Ill.

Beltone Mono-Pac ..... JAMA 130:637 (March 9) 1946
Beltone Harmony Mono-Pac ..... JAMA 133:543 (Feb. 22) 1947
Beltone Symphonette ..... JAMA 140:1217 (Aug. 13) 1949
Beltone Mono-Pac Model M ..... Report not yet published

MFR. BELTONE HEARING AID CO.
1450 W. Nineteenth St., Chicago 8, Ill.

Cleartone Model 500 ..... Report not yet published
Cleartone Regency Model ..... Report not yet published

MFR. AMERICAN SOUND PRODUCTS, INC.
2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model No. 1 ..... JAMA 137:1534 (Aug. 21) 1948

MFR. DYNAMIC HEARING AIDS, INC.
1042 Atlantic Ave., Brooklyn 16, N. Y.

Electroear Model C ..... JAMA 136:769 (March 13) 1948

MFR. AMERICAN EARPHONE CO., INC.
10 E. Forty-third St., New York 17, N. Y.

Gem Model V-35 ..... JAMA 139:229 (Jan. 22) 1949
Gem Model V-60 ..... Report not yet published

MFR. GEM EAR PHONE CO., INC.
50 W. Twenty-ninth St., New York 1, N. Y.

Maico Type K ..... JAMA 129:32 (Sept. 1) 1945
Maico Atomeer ..... JAMA 133:542 (Feb. 22) 1947
Maico UE Atomeer ..... JAMA 143:1069 (July 22) 1950

MFR. MAICO CO., INC.
21 N. Third St., Minneapolis 1, Minn.

Mears Aurophone Model 200 ..... JAMA 138:428 (Oct. 9) 1948
1947-Mears Aurophone Model 98 ..... JAMA 137:1535 (Aug. 21) 1948

MFR. MEARS RADIO HEARING DEVICE CORP.
1 W. Thirty-fourth St., New York 1, N. Y.

Micronic Model 101 (Magnetic Receiver) ..... JAMA 139:99 (Jan. 8) 1949
Micronic Model 303 ..... JAMA 142:1363 (April 29) 1950

MFR. MICRONIC CO.
727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic ..... JAMA 138:1229 (Dec. 25) 1948
Microtone T-4 Audiomatic ..... JAMA 136:109 (Jan. 10) 1948
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